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Diseases of the Eye

Sir Stewart Duke-Elder

Fifteenth Edition

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DISEASES OF THE EYE

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DISEASES OF THE EYE

FIFTEENTH EDITION

BY

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With 21 Coloured Plates and 453 Text figures



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PREFACE TO THE FIFTEENTH EDITION

IN this, the fifteenth edition of this book, the same general philosophy has been retained so that it may serve as a useful guide for the student, the general practitioner and the junior ophthalmic surgeon. An attempt has been made to discuss the commoner ocular diseases at some length, and at the same time make mention of rarer conditions which occasionally present difficulties in diagnosis as well as those diseases which, although rare in temperate countries, assume great importance in tropical areas.

The constant advances of ophthalmic practice in the five years since the last edition appeared have necessitated a large number of alterations; apart from the sections on anatomy and optics, practically every page has required emendation. Recent reorientations of thought on such problems as glaucoma and the retinopathies and others have necessitated considerable alterations. Recent developments in surgery such as the rucking techniques for the treatment of retinal detachment and the use of cryosurgery have also received attention. Some of the illustrations have also been changed and improved.

STEWART DUKE-ELDER.

London, 1969.

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SECTION I

ANATOMY AND PHYSIOLOGY

CHAPTER I

EMBRYOLOGY AND ANATOMY

EMBRYOLOGY

A SHORT general outline of the embryology of the eye is essential to appreciate its anatomy and understand much of its pathology.

The central nervous system is developed from the neural groove which invaginates to form the neural tube running longitudinally down the dorsal surface of the embryo. At either side of the anterior portion of this structure a thickening appears at an early stage (the *optic plate*) which grows outwards towards the surface to form the *primary optic vesicle* (Figs. 1 and 2). From this pair of diverticula from the sides of the forebrain and the mesodermal and ectodermal structures in contact with it the two eyes develop.

After it meets the surface ectoderm, the primary optic vesicle invaginates from below in much the same way as a punctured rubber ball may be pushed into a cup (the *optic cup*), the line of invagination remaining open for some time as the embryonic fissure (Fig. 3). The inner layer of the cup forms the main structure of the retina, the nerve fibres from which eventually grow backwards towards the brain. Its outer layer remains as a single layer of pigmentary epithelium ; between the two lies a narrow space representing the original optic vesicle ; and from its anterior border develop parts of the ciliary body and iris (Fig. 5). Meantime, at the point where the neural ectoderm meets the surface ectoderm, the latter thickens to form the *lens plate*, invaginates to form the *lens vesicle*, and then separates to form the lens ; and through the embryonic fissure the hyaloid artery enters the optic cup and grows forwards to meet the lens, bringing temporary nourishment to the developing structures before it eventually atrophies and disappears ; as it does so, its place is taken by a clear jelly (the vitreous) largely secreted by the surrounding neural ectoderm. While these ectodermal events are happening, the mesoderm surrounding the optic cup differentiates to form the coats of the eye and the orbital structures ; that between the lens and the surface ectoderm becomes hollowed to form the anterior chamber, lined by mesodermal condensations forming the

anterior layers of the iris, the angle of the anterior chamber and the main structures of the cornea ; while the surface ectoderm remains

FIGS. 1 TO 6. THE DEVELOPMENT OF THE EYE.

In each case the solid black is the neural ectoderm, the hatched layer the surface ectoderm and its derivatives, the dotted area is mesoderm ; *a*, cavity of the forebrain ; *b*, cavity of the optic vesicle ; *c*, cavity of the optic cup (or secondary optic vesicle) formed by invagination.

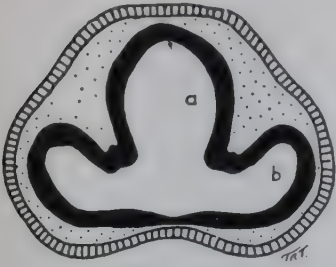


FIG. 1.

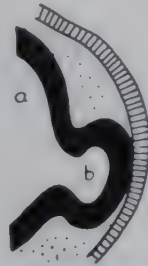


FIG. 2.



FIG. 3.



FIG. 4.

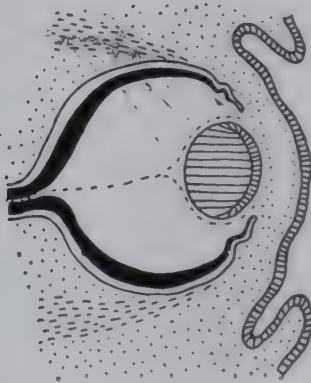


FIG. 5.

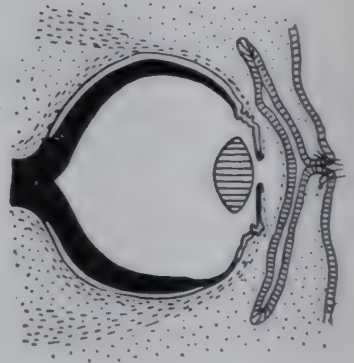


FIG. 6.

FIG. 1. Transverse section through the anterior part of forebrain and optic vesicles of a 4-mm. human embryo.

FIG. 2. The primary optic vesicle.

FIG. 3. The formation of the optic cup by invagination at the embryonic fissure ; invagination of the surface epithelium.

FIG. 4. The optic cup and lens vesicle.

FIG. 5. The formation of the ciliary region and iris, the anterior chamber, the hyaloid artery and the lid folds. The lens is formed from the posterior cells of the lens vesicle.

FIG. 6. The completed eye.

as the corneal and conjunctival epithelium. In the surrounding region folds grow over in front of the cornea, unite, and separate again to form the lids (Figs. 5 and 6).

ANATOMY

The wall of the globe is composed of a dense, imperfectly elastic supporting membrane (Fig. 7). The anterior part of the membrane is transparent—the cornea ; the remainder is opaque—the sclera.

The anterior part of the sclera is covered by mucous membrane—the conjunctiva—which is reflected from its surface onto the lids.

The *cornea* consists of three layers: the epithelium, the substantia propria or stroma, and Descemet's membrane with its endothelium. The epithelium, which is stratified, may be regarded as the continuation of the conjunctiva over the cornea. Its basal membrane lies on a lamina of the substantia propria, called Bowman's membrane. The substantia propria may be regarded as the continuation forwards of the sclera. Descemet's membrane is a

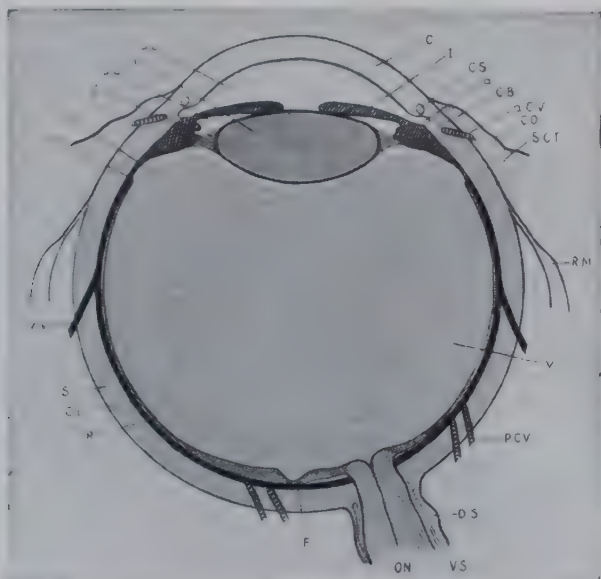


FIG. 7. Diagram of a longitudinal section of the eyeball: a, angle of anterior chamber; a.c., anterior chamber; a.C.V., anterior ciliary vessel; C, cornea; C.B., ciliary body; Ch, choroid; C.O., ocular conjunctiva; C.S., canal of Schlemm; D.S., dural sheath; F, fovea; I, iris; L, lens; O.N., optic nerve; O.S., ora serrata; P.C., posterior chamber; P.C.V., posterior ciliary vessel; P.P., pars plana; R, retina; R.M., rectus muscle; S, sclera; S.C.T., sub-conjunctival tissue; V, vitreous; V.S., vaginal sheath; V.V., vortex vein; Z, zonule.

thin elastic membrane, covered on its posterior surface by endothelium: it may be regarded as the functional continuation forwards of the uveal tract. We shall see that the relationship of the three layers is of some pathological importance, for when, as is often the case, the cornea is affected secondarily by some conjunctival complaint the epithelium and superficial layers are most likely to be affected: similarly, in diseases of the sclera the substantia propria suffers most, and in diseases of the uveal tract, the endothelium, Descemet's membrane and the adjoining posterior layers of the substantia propria.

The cornea is set into the sclera like a watch glass so that the latter overlaps the cornea all round the periphery; the junction of the two tissues is known as the *limbus*. The cornea is very richly

supplied with nerve fibres derived from the trigeminal. It has no blood vessels with the exception of minute arcades, about 1 mm. broad, at the limbus so that it is dependent for its nourishment upon diffusion of tissue-fluid from the vessels at its periphery and materials from the aqueous humour.

Lining the inner aspect of the sclera are two membranes : the highly vascular uveal tract concerned chiefly in the nutrition of the eye, and within this a nervous layer, the true visual nerve ending concerned in the reception and transformation of light stimuli, called the retina.

The uveal tract consists of three parts, of which the two posterior, the choroid and ciliary body, line the sclera while the anterior forms a free circular diaphragm, the iris. The plane of the iris is approxi-

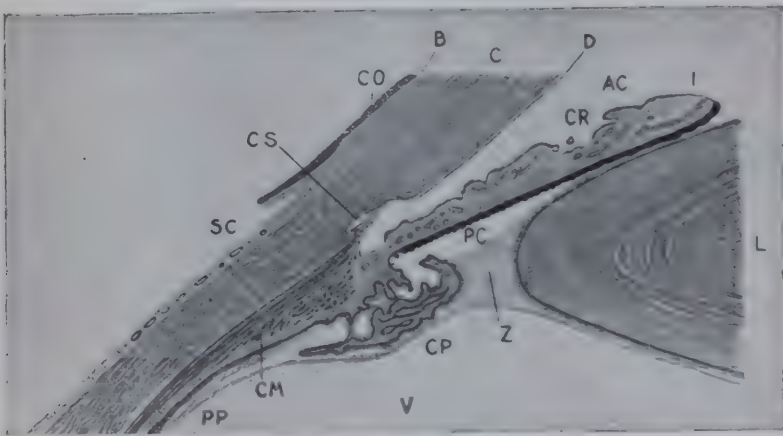


FIG. 8. The region of the angle of the anterior chamber. AC, anterior chamber; B, Bowman's membrane; C, cornea; CM, ciliary muscle; CO, corneal epithelium; CP, ciliary processes; CR, iris crypts; CS, canal of Schlemm; D, Descemet's membrane; I, iris; L, lens; PC, posterior chamber; PP, pars plana of ciliary body; SC, sclera; V, vitreous; Z, zonule of Zinn.

mately coronal : the aperture of the diaphragm is the pupil. Situated behind the iris and in contact with the pupillary margin is the crystalline lens.

The *anterior chamber* is a space filled with fluid, the *aqueous humour* ; it is bounded in front by the cornea, behind by the iris and the part of the anterior surface of the lens which is exposed in the pupil. Its peripheral recess is known as the *angle of the anterior chamber*, bounded posteriorly by the root of the iris and the ciliary body and anteriorly by the corneo-sclera (Fig. 8). In the inner layers of the sclera at this part there is a circular venous sinus, sometimes broken up into more than one lumen, called the *canal of Schlemm*, of great importance in the drainage of the aqueous humour. At the periphery of the angle between the canal of Schlemm and the recess of the anterior chamber there lies a loosely

constructed meshwork of tissues, the *corneo-scleral trabeculæ*. This has a general triangular shape, the apex arising from the termination of Descemet's membrane and the subjacent fibres of the corneal stroma and its base merging into the tissues of the ciliary body and the root of the iris. It is made up of circumferentially disposed flattened bands each perforated by numerous oval stomata through which tortuous passages exist between the anterior chamber and Schlemm's canal in the inner wall of which they open as pores. We shall see that a thorough knowledge of the anatomy of the angle of the anterior chamber is essential for a proper understanding of several pathological problems, especially that of glaucoma.

The anterior chamber is about 2.5 mm. deep in the centre in the normal adult : it is shallower in very young children and also in old people.

The *iris* is thinnest at its attachment to the ciliary body, so that if torn it tends to give way in this region (Fig. 8). It is composed of a stroma containing branched connective tissue cells, usually pigmented but largely unpigmented in blue irides, and with a rich supply of blood vessels which run in a general radial direction. The tissue-spaces communicate directly with the anterior chamber through crypts found mainly near the ciliary border ; this allows the easy transference of fluid between the iris and the anterior chamber. The stroma is covered on its posterior surface by two layers of pigmented epithelium, which developmentally are derived from the retina and are continuous with each other at the pupillary margin. The anterior layer consists of flattened cells, the posterior of cubical cells, and from the epithelial cells of the former two unstriated muscles are developed which control the movements of the pupil, the *sphincter pupillæ*, a circular bundle running round the pupillary margin, and the *dilatator pupillæ*, arranged radially near the root of the iris.

The anterior surface of the iris is covered with a single layer of endothelium, except at some minute depressions or crypts which are found mainly at the ciliary border; it usually atrophies in adult life.

The iris is richly supplied by sensory nerve fibres derived from the trigeminal, a fact which it is important to remember since touching or cutting the iris, especially if it is inflamed, is intensely painful. The *sphincter pupillæ* is supplied by motor nerve fibres derived from the oculomotor nerve, whilst the motor fibres of the *dilatator* muscle are derived from the cervical sympathetic chain.

The *ciliary body* in antero-posterior section is shaped roughly like an isosceles triangle, with the base forwards. The iris is attached to about the middle of the base, so that a small portion of the ciliary body enters into the posterior boundary of the anterior chamber at the angle (Fig. 8). The chief mass of the ciliary body is composed of unstriated muscle fibres, the *ciliary muscle*. This consists of three

parts with a common origin in the ciliary tendon, a structure which runs circumferentially round the globe blending with the "spur" of the sclera and related to the corneo-scleral trabeculæ. The greater part of the muscle is composed of meridional fibres running antero-posteriorly on the inner aspect of the sclera to find a diffuse insertion into the suprachoroid. Most of the remainder of the fibres run so obliquely in interdigitating V-shaped bundles as to give the impression of running in a circle round the ciliary body concentrically with the base of the iris. The third portion of the muscle is composed of a few tenuous iridic fibres arising most internally from the common origin and finding insertion in the root of the iris just anterior to the pigmentary epithelium in close relation to the dilatator muscle.

The inner surface of the ciliary body is divided into two regions ; the anterior part is corrugated with a number of folds running in an antero-posterior direction while the posterior part is smooth. The anterior part is, therefore, called the *pars plicata*, the posterior, the *pars plana*. About 70 plications are visible around the circumference macroscopically, but if microscopical sections are examined, many smaller folds, the *ciliary processes*, will be seen between them. These contain no part of the ciliary muscle, but consist essentially of tufts of blood vessels, not unlike the glomeruli of the kidney. They are covered upon the inner surface by two layers of epithelium, which belong properly to the retina, and are continuous with the similar layers in the iris ; the outer layer, corresponding to the anterior in the iris, consists of flattened cells, the inner of cubical cells, but unlike the condition in the iris, only the outer layer in the ciliary body is pigmented.

The ciliary body extends backwards as far as the *ora serrata*, at which point the retina proper begins abruptly ; the transition from ciliary body to choroid, on the other hand, is gradual, although this line is conveniently accepted as the limit of the two structures. The *ora serrata* thus circles the globe, but is slightly more anterior on the nasal than on the temporal side.

The ciliary body is richly supplied with sensory nerve fibres derived from the trigeminal, so that great pain results from injury or acute inflammation. The ciliary muscle is supplied with motor fibres from the oculomotor and sympathetic nerves.

The *choroid* is an extremely vascular membrane in contact everywhere with the sclera, although not firmly adherent to it so that there is a potential space between the two structures—the *epichoroidal space* (Fig. 9). On the inner side, the choroid is covered by a thin elastic membrane, the *lamina vitrea*, or *membrane of Bruch*. The blood vessels of the choroid increase in size from within outwards, so that immediately beneath the membrane of Bruch there is a capillary plexus, the *choriocapillaris*. Following upon this is the layer of medium-sized vessels, while most externally are the large

vessels, the whole being held together by a stroma consisting of branched pigmented connective tissue cells.

The choroid is supplied with sensory nerve fibres from the tri-

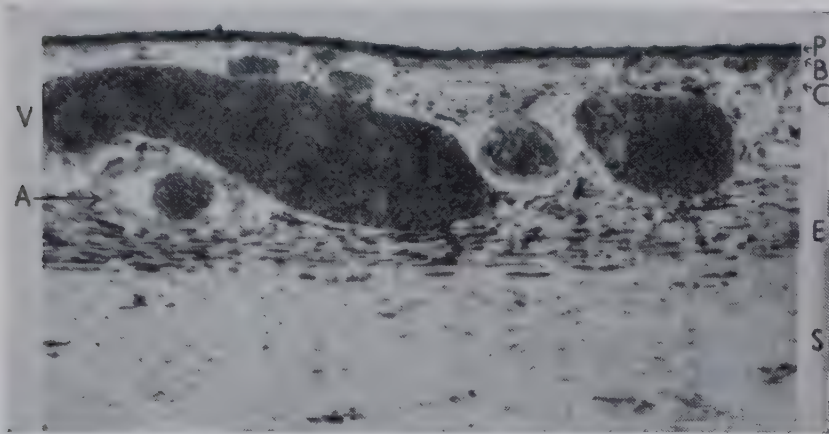
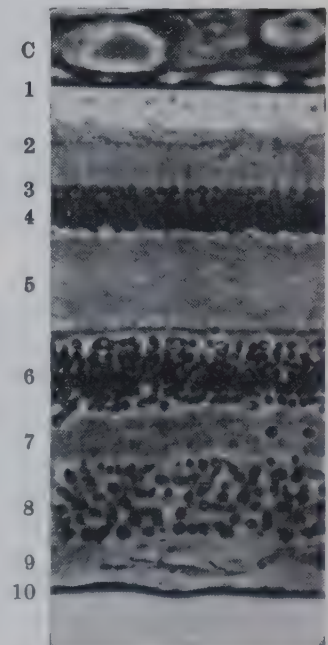


FIG. 9. Transverse section of the choroid near the equator. A, artery; B, Bruch's membrane; C, choriocapillaris; E, epichoroid; P, pigment epithelium; S, sclera; V, vein.

geminal as well as autonomic nerves presumably of vasomotor function.

The *retina* corresponds in extent to the choroid, which it lines, although, as we have seen, the same embryological structure is continued forwards as a double layer of epithelium as far as the pupillary margin. If the two layers of epithelium are traced backwards, the anterior layer in the iris is found to be continuous with

FIG. 10. Section through the parafoveal region of the human retina. C, choroid; 1, pigment epithelium; 2, layer of rods and cones; 3, external limiting membrane; 4, outer nuclear layer; 5, outer plexiform layer; 6, inner nuclear layer; 7, inner plexiform layer; 8, ganglion cell layer; 9, optic nerve fibre layer; 10, internal limiting membrane.



the outer layer in the ciliary body, and this again is continued into the pigment epithelium of the retina, a single layer of hexagonal cells lying immediately adjacent to the membrane of Bruch. Similarly, the posterior layer in the iris, although pigmented, passes into the inner unpigmented layer of the ciliary body, and this suddenly changes at the ora serrata into the highly complex visual retina.

The retina consists of a number of layers formed by three strata of cells and their synapses—the visual cells (lying externally), a relay layer of bipolar cells (lying intermedially), and a layer of ganglion cells (lying internally) the axons of which run into the central nervous system.

Most externally, in contact with the pigment epithelium, is a neural epithelium, the rods and cones which are the end-organs of vision (Fig. 10). Following this, in order from without inwards lie the outer nuclear layer (the nuclei of the rods and cones), the outer plexiform layer comprised of synapses, the inner nuclear layer (the nuclei of the bipolar cells), the inner plexiform layer (again synaptic), the ganglion cell layer, and finally (lying innermost), the nerve-fibre layer composed of the axons of ganglion cells running centrally into the optic nerve. These special nervous constituents are bound together by neuroglia, the better developed vertical cells being called the fibres of Müller, which in addition to acting as a supportive framework, have a nutritive function. The structure is completed by two limiting membranes, the outer perforated by the rods and cones, and the inner separating the retina from the vitreous.

It will be realized that to excite the rods and cones incident light has to traverse the tissues of the retina; it is to be remembered, however, that this arrangement which may superficially look peculiar, allows these visual elements to approximate the (opaque) pigmented layer and their source of nourishment in the chorio-capillaris.

At the posterior pole of the eye, which is situated about 3 mm. to the temporal side of the optic disc, a specially differentiated spot is found in the retina, the *fovea centralis* (Fig. 11). As its name implies,



FIG. 11. The fovea of a primate.

it is a depression or pit, and here only cones are present in the neuro-epithelial layer and the other layers are almost completely absent. The fovea is the most sensitive part of the retina, and it is surrounded by a small area, the *macula lutea*, or yellow spot which, although not so sensitive, is more so than other parts of the retina. It is here that the nuclear layers become gradually thinned out, while, on the other hand, parts of the plexiform layers are especially in evidence: the ganglion cells too, instead of consisting of a single row of cells, are heaped up into several layers. There are no blood vessels in the retina at the macula, so that its nourishment is entirely dependent upon the choroid.

At the optic disc the fibres of the nerve-fibre layer pass into the *optic nerve* (p. 30), the other layers of the retina stopping short abruptly at the edge of the aperture in the scleral canal. This is spanned by a transverse network of connective tissue fibres containing much elastic tissue, the *lamina cribrosa*, through the meshes of which the optic nerve fibres pass; on the posterior side they suddenly become surrounded by medullary sheaths. These fibres, the axons of the ganglion cells of the retina, are, of course, afferent or centripetal fibres, but the optic nerve also contains a few efferent or centrifugal fibres.

The *lens* is a biconvex mass of peculiarly differentiated epithelium. It will be remembered that it is developed from an invagination of the epidermal epiblast of the foetus, so that what was originally the surface of the epithelium comes to lie in the centre of the lens, the peripheral cells corresponding to the basal cells of the epidermis. Just as the epidermis grows by the proliferation of the basal cells, the old superficial cells being cast off, so the lens grows by the proliferation of the peripheral cells. The old cells, however, cannot be cast off, but undergo changes (sclerosis) analogous to that in the stratum granulosum of the epidermis, and become massed together in the centre or nucleus: moreover, the newly formed cells elongate into fibres, the lens fibres, which have a complicated architectural form, being arranged in zones wherein the fibres growing from opposite directions meet in sutures. Without going into details, it is important to bear in mind that the central nucleus of the lens consists of the oldest cells and the periphery or cortex of the youngest (Fig. 12). The fibres of the central *embryonic nucleus* meet around Y-shaped

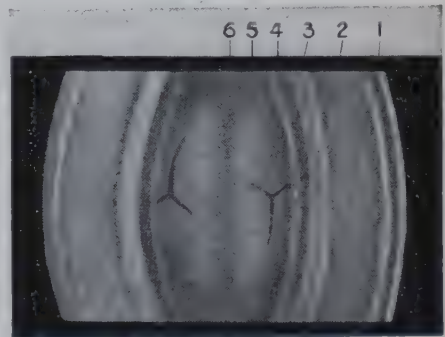


FIG. 12. The structure of the lens in an adult of forty years, as shown in the optical beam of the slit-lamp. 1, anterior capsule; 2, cortex; 3, adult nucleus; 4, infantile nucleus; 5, foetal nucleus; 6, embryonic nucleus.

sutures. Outside this embryonic nucleus, successive nuclear zones are laid down as development proceeds, called, depending on the period of formation, the *fœtal nucleus* (corresponding to the lens at birth), the *infantile nucleus* (corresponding to the lens at puberty), the *adult nucleus* (corresponding to the lens in early adult life), and finally and most peripherally, the *cortex* comprised of the youngest fibres. In this part of the lens also the fibres meet along sutures with a general stellate arrangement. The mass of epithelium which constitutes the lens is surrounded by a hyaline membrane, the *lens capsule*, which is thicker over the anterior than over the posterior surface (p. 63); it is a cuticular deposit secreted by the epithelial cells having on the outside a thin membrane, the *zonular lamella*.

The lens in fœtal life is almost spherical; it gradually becomes flattened so as to assume a biconvex shape. It is held in place by the *suspensory ligament* or *zonule of Zinn*. This is not a complete membrane, but consists of bundles of strands which pass from the surface of the ciliary body to the capsule where they join with the zonular lamella. The strands pass in various directions so that the bundles often cross one another. Thus the most posterior arise from the pars plana of the ciliary body almost as far back as the ora serrata; these lie in contact for a considerable distance with the ciliary body and then curve towards the equator of the lens to be inserted into the capsule slightly anterior to the equator. A second group of bundles springs from the summits and sides of the ciliary processes, i.e., far forwards, and passes backwards to be inserted into the lens capsule slightly posterior to the equator. A third group passes from the summits of the processes almost directly inwards to be inserted at the equator.

It will be noticed that there is a somewhat triangular space between the back of the iris and the anterior surface of the lens, having its apex at the point where the pupillary margin comes in contact with the lens; it is bounded on the outer side by the ciliary body. This is the *posterior chamber* and contains aqueous humour.

Behind the lens is the large vitreous chamber, containing the *vitreous humour*. This is a jelly-like material chemically of the nature of an inert gel containing a few cells and wandering leucocytes. The fibres seen in histological sections are probably artefacts except in pathological conditions. As in other gels the concentration of the micellæ on the surface gives rise to the appearance of a boundary membrane in sections—the so-called *hyaloid membrane*.

THE BLOOD SUPPLY OF THE EYE

The arrangement of the blood vessels which supply the eye is peculiar, and is of great importance in considering pathological conditions.

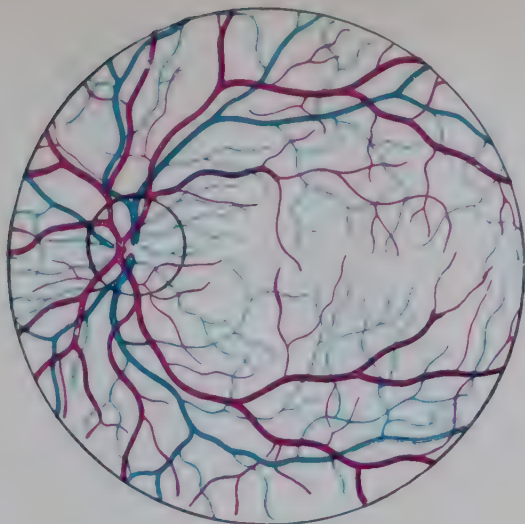


FIG. 1. The retinal circulation.

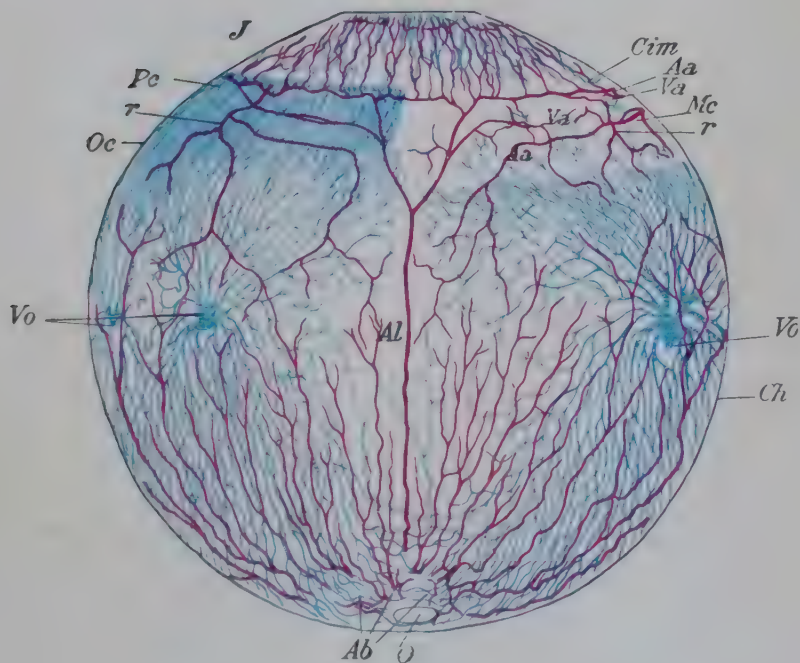
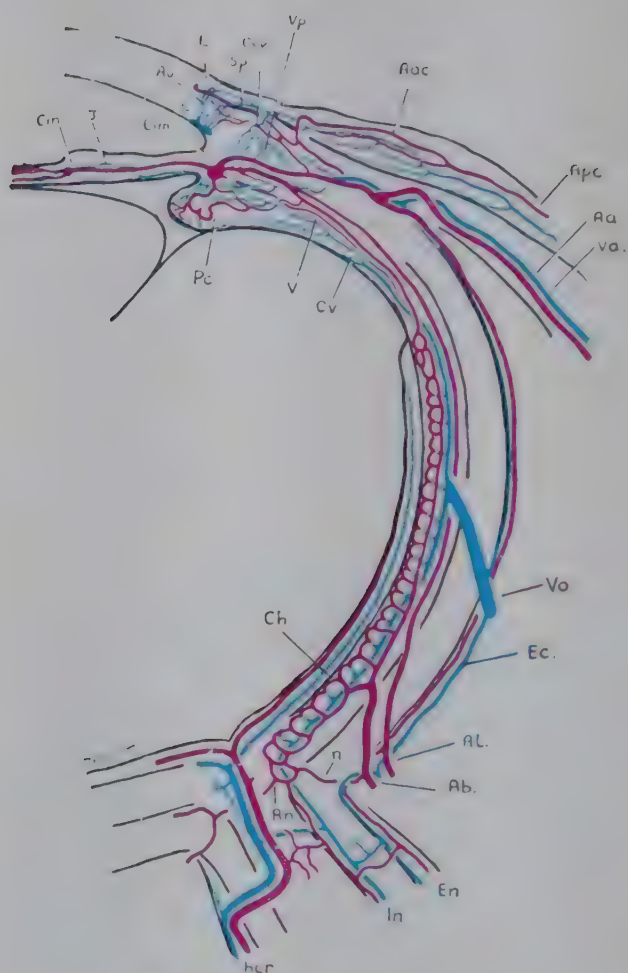


FIG. 2. The choroidal circulation.

Aa, anterior ciliary arteries; Ab, short posterior ciliary arteries; Al, long posterior ciliary arteries; Ch, choroid; Cim, circulus iridis major; J, vessels of the iris; Mc, ciliary muscle; O, optic nerve; Oc, orbiculus ciliaris; Pc, ciliary processes; r, recurrent ciliary arteries; Va, anterior ciliary veins; Vo, vortex vein.



THE CILIARY CIRCULATION

Aa, anterior ciliary artery : Aac, anterior conjunctival artery : Ab, short posterior ciliary artery : Acr, central artery of the retina : Al, long posterior ciliary artery : An, anastomosis of choroidal vessels with those of the optic nerve : Apc, posterior conjunctival artery : Av, aqueous vein : C, canal of Schlemm : Cev, ciliary efferent vein : Ch, choriocapillaris : Cim, circulus iridis major : Cin, circulus iridis minor : Cv, recurrent choroidal veins : Ec, episcleral artery : En, vessels to the outer sheath of the optic nerve : J, radial vessels of iris : In, vessels to the inner sheath of the optic nerve : L, limbal vessels : n, branch or short posterior ciliary artery to optic nerve : Pc, ciliary plexus : Sp, scleral plexus : V, branch of vena vorticiosa from ciliary system : Va, anterior ciliary vein : Vo, vortex vein : Vp, ciliary venous plexus.

The arteries of the eye in man are all derived from the ophthalmic artery, which is a branch of the internal carotid. The ophthalmic artery has few anastomoses, so that on the arterial side the ocular circulation is an offshoot of the intracranial circulation. This does not apply in so marked a degree to the venous outflow from the eye. While in man most of the blood passes to the cavernous sinus by way of the ophthalmic veins, it must be remembered that these anastomose freely in the orbit, the superior ophthalmic vein communicating with the angular vein at the root of the nose, and the inferior ophthalmic vein with the pterygoid plexus. Hence too great stress must not be laid upon the venous circulation in the retina as a guide to the condition of the intracranial circulation.

The retina is supplied by the central artery, which enters the nerve on its lower surface, 15–20 mm. behind the globe. The central artery divides on or slightly posterior to the surface of the disc into the main retinal trunks, which will be considered in detail later (Plate I). The retinal arteries are end-arteries and have no anastomoses at the ora serrata. The only place where the retinal system anastomoses with any other is in the neighbourhood of the lamina cribrosa. The veins of the retina do not accurately follow the course of the arteries, but they behave similarly at the disc, uniting on or slightly posterior to its surface to form the central vein of the retina, which follows the course of the corresponding artery.

The uveal tract is supplied by the ciliary arteries, which are divided into three groups—the short posterior, the long posterior, and the anterior (Plates I and II). The short posterior ciliary arteries, about twenty in number, pierce the sclera in a ring around the optic nerve, running perpendicularly through the sclera, to which fine branches are given off. The long posterior ciliary arteries, two in number, pierce the sclera slightly farther away from the nerve in the horizontal meridian, one on the nasal, the other on the temporal side. They traverse the sclera very obliquely, running in it for a distance of 4 mm. Both these groups are derived from the ophthalmic artery, while the anterior ciliary arteries are derived from the muscular branches of the ophthalmic artery to the four recti. They pierce the sclera 5 or 6 mm. behind the limbus or corneo-scleral junction, giving off twigs to this region to the conjunctiva, the sclera and the anterior part of the uveal tract.

The ciliary veins also form three groups—the short posterior ciliary, the *venæ vorticosæ*, and the anterior ciliary. The short posterior ciliary veins are unimportant; they do not receive any blood from the choroid, but only from the sclera. The *venæ vorticosæ* are the most important, consisting usually of four large trunks which open into the ophthalmic veins. They enter the sclera slightly behind the equator of the globe, two above and two below, and pass very obliquely through this tissue. The anterior ciliary

veins are smaller than the corresponding arteries, since they receive blood from only the outer part of the ciliary muscle.

Of these ciliary vessels the short posterior ciliary arteries supply the whole of the choroid, being reinforced anteriorly by anastomoses with recurrent branches from the ciliary body. The ciliary body and iris are supplied by the long posterior and anterior ciliary arteries. The blood from the whole of the uveal tract, with the exception of the outer part of the ciliary muscle, normally leaves the eye by the *venæ vorticosæ* only.

The two long posterior ciliary arteries pass forwards between the choroid and the sclera, without dividing, as far as the posterior part of the ciliary body. Here each divides into two branches (Plate II):

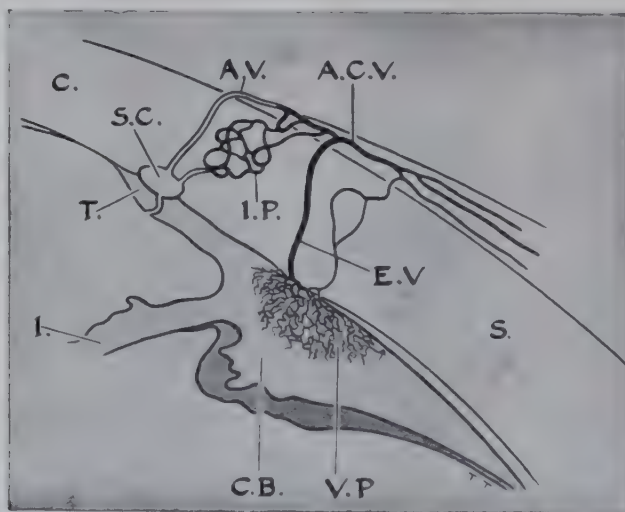


FIG. 13. The exit channels of the aqueous humour in man. C, cornea ; S, sclera ; I, iris ; CB, ciliary body. The primitive drainage channels of lower animals are seen in VP, the ciliary venous plexus, draining by EV, the ciliary efferent veins into ACV, the anterior ciliary veins. Superimposed on this is the drainage system peculiar to primates, represented by T, the trabeculae, SC, the canal of Schlemm, IP, the intrasccleral plexus, and AV, an aqueous vein emptying into the anterior ciliary veins.

they run forward in the ciliary muscle, and at its anterior part bend round in a circular direction, anastomosing with each other and thus forming the *circulus arteriosus iridis major*. This is situated in the ciliary body at the base of the iris : from it the ciliary processes and iris are supplied. Other branches from the major arterial circle run radially through the iris, dividing dendritically and ending in loops at the pupillary margin. A circular anastomosis takes place a little outside the pupillary margin, the *circulus arteriosus iridis minor*.

The tributaries of the vortex veins, which receive the whole of the blood from the choroid and iris, are arranged radially, the radii being bent, so as to give a whorled appearance—hence their name.

The veins of the iris are collected into radial bundles which pass backwards through the ciliary body, receiving tributaries from the ciliary processes. Thus reinforced, they form an immense number of veins running backwards parallel to each other through the smooth part of the ciliary body. After reaching the choroid they converge to form the large anterior tributaries of the vortex veins.

The veins from the outer part of the ciliary body, on the other hand, pass forward and unite with others to form a plexus (the *ciliary venous plexus*) which drains into the anterior ciliary veins and the episcleral veins. These vessels communicate directly with

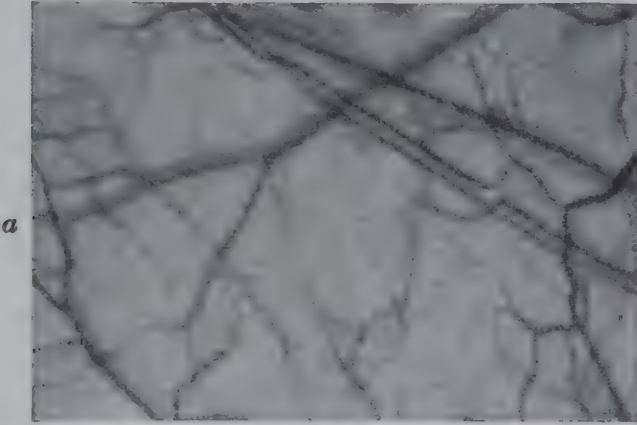


FIG. 14. Photograph of the episcleral circulation to show a laminated aqueous vein (*a*) with clear contents in its upper part and dark blood in its lower part.

the canal of Schlemm which we have seen is in intimate connection with the anterior chamber by means of numerous tortuous channels through the loose tissue of the trabeculæ (Fig. 8). From this canal the efferent channels form a complex system (Fig. 13); some of them drain into efferent ciliary veins in the sclera while others traverse the sclera and only join the venous system in the subconjunctival tissues (*aqueous veins*) (Fig. 14). We shall see that these venous effluents are of great importance in regulating the drainage of the aqueous humour which takes place largely through the canal of Schlemm.

The marginal loops of the cornea and the conjunctival vessels are branches of the anterior ciliary vessels (Plate II).

CHAPTER 2

THE PHYSIOLOGY OF THE EYE

IN order that the eye may satisfactorily perform its duties as an organ of vision it is essential that its media should be optically transparent and that sharp images of objects in the outer world be formed upon the retina. For these purposes the cornea and the lens must be avascular and transparent and the fluid filling the globe be optically clear and at the same time remain under sufficient pressure to keep the walls of the globe taut and the optical surfaces in proper position. To attain these purposes the physiology of the eye differs in many respects from that of other organs, the main problems being concerned with the nature and formation of the intra-ocular fluid and the maintenance of the intra-ocular pressure.

The nature and formation of the intra-ocular fluid are subjects on which much research has been expended. For many years Leber's theory that it was a simple filtration from the blood was generally accepted. At a later date, however, chemical analysis of the aqueous humour showed that such a simple hypothesis could not explain the facts ; but the most recent work undertaken on this difficult subject indicates that two mechanisms are involved—ultrafiltration and secretion.

It is well known that a two-way transference of fluid (tissue-fluid) occurs through the capillary walls in all the organs of the body ; thereby nutriment is conveyed to the tissues and metabolites removed. The capillaries in different tissues vary considerably in their permeability to suit local needs. Those of the liver, for example, are very permeable and a protein-rich milky lymph escapes. For optical purposes those in the eye (like those in the central nervous system) are relatively impermeable so that practically no colloid molecules can pass into the cavity of the eye. A filtrate which is practically protein-free, derived from the plasma, therefore passes freely inwards and outwards between all the uveal or retinal capillaries and the cavity of the eye to form the *intra-ocular fluid*. This occurs throughout the whole eye but, owing to the anatomical configuration of the ciliary body where the enlarged bunches of wide capillaries resemble the glomeruli of the kidney, much of the transference of fluid into the eye occurs in this region.

The system of semi-permeable membranes separating the blood from the ocular cavity is known as the *blood-aqueous barrier*, the composition of which is shown in Fig. 15. It is formed in the pos-

terior segment of the globe by the walls of the retinal capillaries which, like those of the central nervous system are very impermeable. In the ciliary region it is formed by the walls of the uveal capillaries with, in addition, the two-layered ciliary epithelium which the fluid must traverse before the posterior chamber is reached. In the iris it is formed by the walls of the capillaries in this tissue which are freely exposed to the anterior chamber through the crypts and spongy stroma.

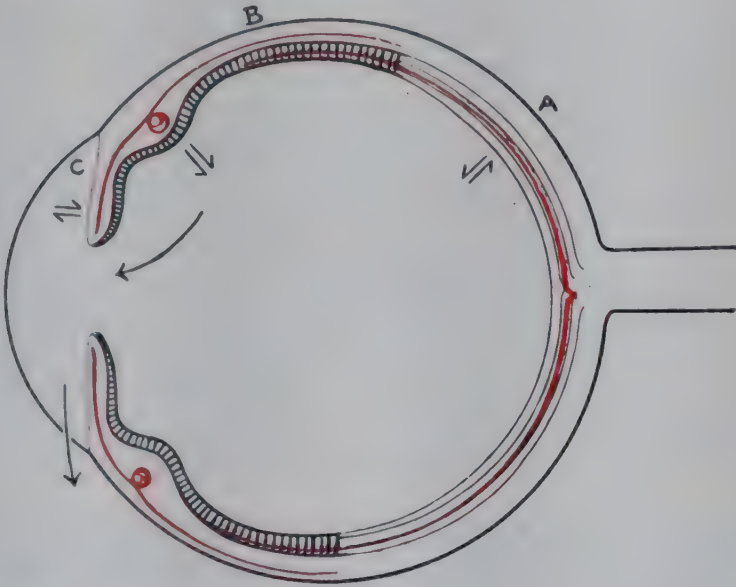


FIG. 15. The effective blood-aqueous barrier. A. In this zone the barrier is formed by retinal capillaries. B. In this zone the barrier is formed partly by retinal capillaries and partly by uveal capillaries plus the ectodermal layer formed by the anterior part of the retina and its prolongation as the ciliary and iris epithelium. C. In this zone the barrier is formed by the uveal capillaries only. The arrows indicate that in A and C, two-way traffic exists; in the ciliary region fluid traffic is essentially into the cavity of the eye, determining a circulation through the pupil and out at the angle of the anterior chamber.

This peculiar impermeability of the ocular capillaries, while necessary from the optical point of view, forbids the ready passage of large-sized molecules of any kind into the eye. Such therapeutic substances as arsenic or penicillin when administered systemically are thus of little value in ocular therapeutics. Substances with a high lipoid-solubility, however, which easily penetrate living cells, traverse the barrier much more readily (sulphonamides, chlor-ampenicol, etc.).

It is obvious that if the permeability of the capillaries is increased, large molecules will be able to pass through their walls, so that a turbid fluid rich in protein is formed—*plasmoid aqueous*. This increase in permeability may be brought about by vasodilator drugs and occurs in inflammatory conditions such as iridocyclitis or

choroiditis, and also if the capillary walls are mechanically stretched by suddenly lowering the intra-ocular pressure and removing their external support. This occurs when the globe is suddenly opened as by paracentesis or a surgical operation.

Such a two-way transference of fluid across the capillary walls would tend to stagnation. To it is added a secretory process conducted by the metabolic activity of the cells of the ciliary epithelium; it is probable that this accounts for some 75 per cent. of the total quantity of aqueous. The intimate mechanism of secretion is not understood, but it is known that a watery fluid rich in sodium and containing small quantities of ascorbic acid and other substances is poured into the posterior chamber.

Having this dual origin, the aqueous humour thus consists of a dilute solution of all the diffusible constituents of the plasma, in addition to the substances specifically secreted. Since entry into the eye across the blood-aqueous barrier is difficult and, as we shall see, exit through the drainage channels is easy, many of the constituents of the aqueous humour are in deficit in comparison with the blood with the exception of those secreted. There is, however, an incidental excess of lactic acid in the aqueous compared with the blood due to the formation of this substance as an end-product of the metabolism of the lens.

A circulation of the aqueous humour is necessary both for metabolic purposes and to regulate the intra-ocular pressure. We have seen that the greater part of the fluid is formed in the ciliary region; it flows from the posterior chamber through the pupil into the anterior chamber and escapes through the drainage channels at the angle, and thence into the episcleral veins. In addition there is a second accessory exit (the *uveo-scleral outflow*) through the ciliary body into the choroid and suprachoroid and thence into the episcleral tissue; although a minor means of exit, this pathway may sometimes be of importance.

The anatomy of the main drainage channels has already been described (Fig. 13). The physiology of the system is seen in Figs. 16 to 18. The crux of the matter lies in the relationship between the intra-ocular pressure and the blood pressure in the veins as they traverse the sclera. Since the walls of the veins are readily compressible, they are at once constricted if the intra-ocular pressure rises above the venous pressure at its lowest point, that is, just as they leave the eye (Fig. 17); the venous pressure behind the constriction banks up until it rises above the intra-ocular pressure and the blood-flow is resumed. For mechanical reasons, therefore, the pressure in the exit veins (at the angle of the anterior chamber, in the *venæ vorticosæ*, and in the veins at the optic disc) normally remains just above the intra-ocular pressure and theoretically will do so until the level of pressure in the ophthalmic artery is reached. This is the highest venous pressure in the body.

In normal circumstances, therefore, the venous pressure just within

the sclera is slightly above the intra-ocular pressure (15–20 mm. Hg) and immediately thereafter there is a sudden drop of pressure in the very short journey taken by the vein as it traverses the sclera and becomes episcleral. The aqueous in the anterior chamber filters readily through the channels of the trabeculae at the angle of the anterior chamber into the canal of Schlemm and thence through its intrascleral effluents and aqueous veins into the venous system at a point beyond this rapid fall of pressure (Fig. 16) ; free drainage is thus secured. It is estimated that approximately 2 c.mm. of fluid, or approximately 1 per cent. of

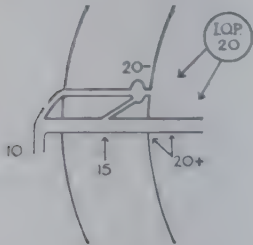


FIG. 16. The fall of pressure in the canal of Schlemm and exit veins in the normal eye.

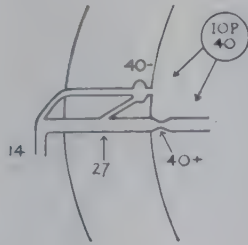


FIG. 17. The fall of pressure on raising the intra-ocular pressure passively.

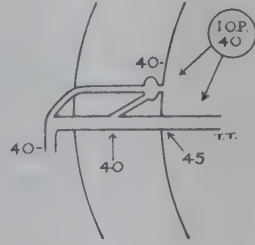


FIG. 18. The pressure when the venous pressure is raised.

the fluid in the anterior chamber drains away in this manner every minute.

The mechanism of drainage in abnormal conditions is important. We have just seen that if the intra-ocular pressure is raised (Fig. 17), the venous pressure just inside the sclera must rise with it ; the considerable fall between this point and the episcleral veins thus becomes accentuated, and drainage is increased. In these circumstances the canal of Schlemm and its exit channels act as an efficient safety-valve, tending to maintain the intra-ocular pressure at its normal level.

If, however, the venous pressure is primarily raised, the pressure throughout the venous exits remains higher than the pressure in the

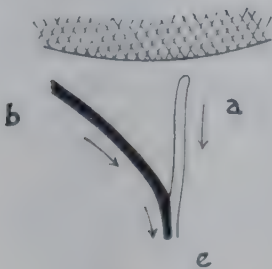


FIG. 19.

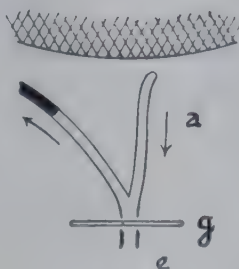


FIG. 20.

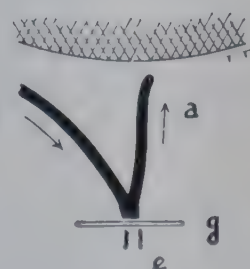


FIG. 21.

FIGS. 19, 20 and 21. The glass-rod phenomenon. In each case *a* represents an aqueous vein, *b* a blood vein, and *e* a recipient vein formed by the junction of the two. The arrows indicate the direction of flow. In Fig. 19, all flow is from the limbus and the recipient vein shows a laminated stream. In Fig. 20 the recipient vein is blocked at *g*, and an aqueous influx takes place into the blood vein. In Fig. 21 a blood influx takes place into the aqueous vein.

anterior chamber and the canal of Schlemm (Fig. 18). In these circumstances drainage is embarrassed or becomes impossible so that the aqueous is banked up until the intra-ocular pressure rises to still greater heights when drainage may again be resumed. As we shall see, therefore, the venous pressure is important in the regulation of the intra-ocular pressure.

These pressure relationships may be visualized in the vessels on the sclera. The veins contain blood, the aqueous veins aqueous, and when two such vessels meet to form a confluent vessel, a laminated stream is often seen (Fig. 14). When such a vessel is compressed, as by a glass rod, fluid flows from whichever vessel is at a higher pressure into the other (the *glass-rod phenomenon*). If the vein is at a higher pressure there is a "blood influx" into the aqueous vein; if the aqueous is at a higher pressure, an "aqueous influx" is seen into the blood vein (Figs. 19-21). The former is seen during a rising phase of tension in glaucoma or when the pressure in the anterior chamber is lowered artificially by a paracentesis, the latter in a falling phase or when external pressure is put upon the globe as by pressing upon the upper lid with the finger.

The Intra-ocular Pressure. We are now in a position to discuss the mechanism of the maintenance of the normal intra-ocular pressure. Prolonged changes are essentially caused by two factors: (1) an alteration in the forces determining the formation of the aqueous, and (2) alterations in the resistance to its outflow. From the clinical point of view the latter is the more important. No instance has been conclusively proved of increased ocular tension being associated with increased secretion, but the action of drugs or the destruction of the ciliary epithelium by inflammatory processes may result in a reduction of secretion and a softening of the eye. A rise in the intra-ocular pressure may be caused either by an increase in the pressure in the episcleral veins into which the aqueous drains or by any process which blocks the seepage of aqueous into the canal of Schlemm, such as sclerosis of the trabeculae or their obstruction by exudates or organized tissues. In either event glaucoma is the result. It is to be remembered, however, that even if the drainage channels to the canal of Schlemm are blocked, the intra-ocular pressure does not rise indefinitely; it cannot rise above the mean blood pressure since at that point the circulation will cease; moreover, some drainage of intra-ocular fluid will take place through the uveo-scleral outflow, but this method of outlet is less effective than the main drainage channels.

While these are the principal factors determining prolonged changes in the intra-ocular pressure, other agencies can exert more temporary effects.

(1) *Variations in the hydrostatic pressure in the capillaries.* It is obvious that the pressure in the eye will follow all such variations; thus it follows faithfully the pulse and respiratory rhythms (Fig. 22). In view of the fact that the entire uveal tract is virtually erectile

tissue, it will be understood that this influence may be considerable. It is important to remember, however, that it is the pressure in the capillaries, not the general blood-pressure, that matters. Experimentally sudden changes in the general blood-pressure usually break through to the capillaries so that changes in the former are reflected in the intra-ocular pressure (stimulation of the



FIG. 22. The normal intra-ocular pressure (dog). Upper curve—blood pressure (mercury manometer). Lower curve— intra-ocular pressure (optical manometer). The large waves are respiratory excursions and the small waves are due to pulse beats.

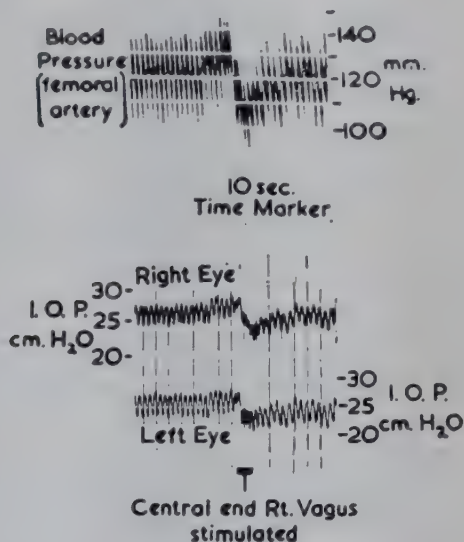


FIG. 23. To show the variation of the intra-ocular pressure and blood pressure. The central end of the right vagus was stimulated in the rabbit. Upper curve, the blood pressure in millimetres of mercury; the lower two curves, the intra-ocular pressures taken by an optical manometer and calibrated in centimetres of water. The fall in the intra-ocular pressure on the right (stimulated side) is seen to be greater than that on the left.

vasomotor centre, sympathetic nerves, injection of adrenaline, nicotine, etc.) (Fig. 23) : but in clinical conditions a high blood-pressure is usually banked down by arteriolar constriction so that the pressure in the eye does not follow that in the main arteries of the body, and a raised ocular tension is not associated with hypertensive states.

In general it may be said that if arteriolar dilatation allows more blood at a higher pressure to enter the capillary bed a higher tension is created in the eye provided the capillaries maintain their *status quo*. If capillary dilatation occurs as a separate phenomenon with-

out alteration in the feeding arterioles, a low hydrostatic pressure results in the widely dissipated capillary bed ; the volumetric change involved is readily compensated in the normal eye through the drainage channels. If both arteriolar and capillary dilatation occur, the resultant change will depend on whichever of the two influences predominates. If, however, obstruction occurs in the venous system, the rise of pressure therein will readily become effective in the capillaries and a rise in intra-ocular pressure will result. It is important to realize that a vasodilatation does not necessarily lead to an increased intra-ocular pressure ; the contrary is very frequently the case.

(2) *An increase in permeability of the capillaries*, allowing the formation of a plasmoid aqueous with a high protein content, will increase its osmotic pressure relatively to that of the blood and thus raise the pressure in the eye, a process accentuated if the drainage channels become clogged. As we have seen this occurs particularly in inflammations.

(3) *A change in the osmotic pressure of the blood* will be reflected in the intra-ocular pressure by altering the process of diffusion across the capillary walls, hypotonicity inducing a rise and hypertonicity a fall. This can be demonstrated experimentally, but in clinical conditions such changes are small and gradual in their development so that, damped down in degree and time-relations by the relative impermeability of the blood-aqueous barrier and compensated by other factors, they are rarely effective except in extreme conditions such as diabetic coma.

(4) *Volumetric changes* within the globe should be immediately transformed into pressure changes owing to the indistensibility of the sclera ; if extra fluid, for example, were forced into the eye its tension should rise abruptly. The safety-valve action of the exit channels, however, normally compensates for such changes by allowing a corresponding loss of aqueous. To be effective they must therefore be considerable and abrupt, such as a large increase in blood-volume owing to sudden vasodilatation ; and even then they are transient. The maintenance of a normal intra-ocular pressure in the presence of a large tumour indicates the efficiency of the compensatory mechanism in maintaining the pressure of the eye by the expulsion of fluid despite gross volumetric changes.

(5) *A blockage of the circulation of aqueous*, on the other hand, has a profound effect in raising the ocular tension. Such a block may occur in two places : (a) at the pupil where the flow of fluid from the posterior to the anterior chamber may be impeded, and (b) at the angle of the anterior chamber.

Obstruction in the first of these situations is usually due to one of two causes. The first arises in eyes with a shallow anterior chamber when the margin of the iris is firmly apposed to the anterior surface of the lens thus causing the condition of " blockage of the pupil "

wherein the aqueous becomes dammed in the posterior chamber; the iris is thus bellied forwards to block the angle of the anterior chamber leading to a primary closed-angle glaucoma. The second is due to organic changes when the iris becomes adherent to the anterior capsule of the lens in inflammatory conditions, when secondary glaucoma occurs. Inefficiency of the drainage channels, on the other hand, as we have seen, causes either a cumulative rise of pressure or transient increments.

The intra-ocular pressure within the eye normally varies from 15 to 20 mm. Hg. It is most accurately measured by a *manometer*, whereby a small cannula is inserted into the anterior chamber connected with a small-bored mercury or saline manometer. Such a technique is used experimentally on animals but its clinical application is obviously impossible. Since, however, the sclera is only very slightly elastic and is rendered tense by the internal pressure, a measurement of the degree to which it can be indented on the application of a standard weight gives an indication of the intra-ocular pressure with considerable accuracy. Such a method is used clinically in *tonometry* (p. 110). The result thus obtained (usually given also as mm. Hg by standardization with a manometer on experimental animals) is referred to as the *ocular tension*.

The Nervous and Pharmacological Control of the Circulation. Why the intra-ocular pressure remains at its normal level is not known with certainty. Evidence is accumulating, however, which is now reasonably conclusive, that an area exists in the hypothalamus which exercises such a control. Here the electrical stimulation of particular regions is followed by either a rise or a fall in the intra-ocular pressure independent of changes in the blood pressure. To this centre the afferent paths from the eye have not been determined; from it the only known path so far demonstrated is through the sympathetic, the fibres travelling down the cervical cord, relaying in the cervical ganglion, and travelling to the eye by way of the cervical chain and the ophthalmic artery. The action of this nerve is vasoconstrictive (adrenergic); vasodilators have not yet been demonstrated but cholinergic drugs have an effective action. It is probable that through the activity of this area the pressure within the eye is maintained at its homeostatic level despite considerable variations in the other factors which tend to alter it. In this connection the occurrence of normal diurnal variations in pressure is important (Fig. 24). The average variation throughout the day in the tension of the eye is about 2 mm. Hg, a rise occurring sometimes in the morning, sometimes in the afternoon, while frequently a biphasic variation occurs. This swing in tension is abolished by blocking the sympathetic nerve supply to the eye in the orbit or the neck and is presumably subject to central control as are the similarly phasic variations of temperature, sleep,

diuresis and other bodily functions. These diurnal variations are important because of their exaggeration in glaucoma.

In addition to this central control, there is a local nervous control by axon reflexes mediated through the fifth nerve. Such reflexes act similarly to the "triple response" described by Lewis in the skin, and are due to the liberation of histamine-like substances. Any local irritation of the iris, mechanical (as by stroking it with a needle) or inflammatory, sets up a vasodilatation over the entire uvea accompanied by an increased permeability of the capillaries and the formation of a plasmoid aqueous. This action is abolished by throwing the fifth nerve-endings out of action, as by cocaine. When we remember that the uveal tract is virtually composed of erectile tissue, it is evident that such a reaction can produce an immediate rise in the ocular tension.

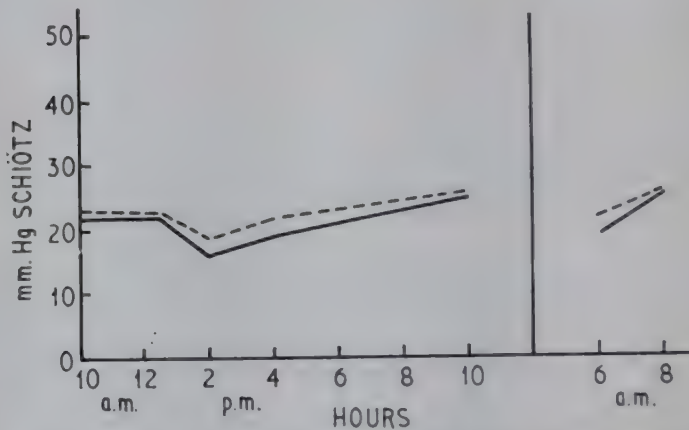


FIG. 24. Normal diurnal variation of the intra-ocular pressure. The abscissæ represent times of day; the vertical line indicates when the subject was asleep. Continuous line, right eye; dotted line, left eye.

Certain drugs affect the intra-ocular pressure. Thus adrenergic drugs (adrenaline, etc.) act by causing a vasoconstriction and diminishing the secretion of the aqueous, and normally lower the ocular tension. Cholinergic drugs, such as eserine or pilocarpine, increase the drainage of the aqueous through the trabeculæ and therefore lower the tension of the eye.

THE METABOLISM OF THE OCULAR TISSUES

The vascularized tissues of the eye, particularly the uveal tract, differ in no respect in their general metabolism from other tissues in the body. This applies also to the vascularized (inner) layers of the retina which correspond to other central nervous tissues; in both these cases the blood vessels are insulated by ectodermal sheaths and the capillaries are relatively impermeable to colloids, salts, and acid dye-stuffs. The outer layers of the retina (rods and cones), on the other hand, are nourished by diffusion from the under-

lying choriocapillaris ; when the choroidal circulation is obstructed, experimentally by cutting the ciliary arteries or clinically in disease, these layers of the retina die. As a whole, the retina has a very active metabolism, comparable indeed to sarcomatous tissue, and if its sources of nutriment are cut off, it rapidly dies.

The non-vascularized tissues of the eye—the cornea and the lens—must obviously have a specialized metabolism, and so far as our present knowledge goes, they depend for their energy requirements essentially on carbohydrates which are utilized by phosphorylation and autoxidative mechanisms.

The **cornea** has few energy requirements which are necessary for the replacement of its tissues and the maintenance of transparency. The latter depends essentially on its state of relative dehydration which is maintained by an active transference of fluid outwards through the epithelium and endothelium, particularly the latter. A fall in metabolic activity or an increase in the permeability of its membranes thus leads to oedema and opacification. The essential physiological differences between the cornea and the sclera are that in the cornea the fibrils are arranged in a regular lattice-work in a ground-substance of mucopolysaccharide whereas the fibres of the sclera are irregularly arranged, and that the former tissue is bounded by cellular membranes by which its fluid-traffic is controlled.

The cornea derives its nutriment from three sources—oxygen directly from the air and solutes from the perilimbal capillaries and the aqueous humour. The first is an active process undertaken by the epithelium, and in an atmosphere of nitrogen, lactic acid collects rapidly in this layer of cells. The importance of diffusion from the limbal capillaries is seen clinically in the relative resistance of the peripheral parts of the cornea to degenerative changes, but at the same time, if these vessels are experimentally cut, corneal transparency is maintained. Similarly, if the aqueous is replaced by nitrogen, the cornea remains transparent ; but it turns opaque if both these sources of nutrition are cut off. Metabolic activity which exhibits a high rate of aerobic glycolysis is maintained by the aid of enzyme systems, as occurs in the lens.

The **lens** derives its nourishment entirely from the aqueous humour in which it is immersed, the fluid-traffic being regulated by the semi-permeability of the capsule and the subcapsular epithelium. If this membrane is disrupted, the whole tissue, like the cornea, tends to adsorb fluid and turn opaque. Active transport takes place between the lens and the aqueous owing to the activity of the subcapsular epithelium and the capsule itself is freely permeable to water and electrolytes as well as colloids of small molecular size, the posterior part being more permeable than the anterior. It is interesting that the permeability of the whole decreases with age.

The fact that the lens has a respiratory quotient (CO_2/O_2) of 1.0 shows that carbohydrate is its essential source of energy, a conclusion

confirmed by the fact that the aqueous in the aphakic eye contains more glucose than in the normal eye. Chemical studies have shown that the initial stage in the break-down of the sugar is its combination with phosphates (phosphorylation) in the production of pyruvic acid ; by experiments with radio-active tracers this has been found to occur particularly in the cortical layers. In all tissues this chemical process is effected by enzymes (such as hexokinase) which have been demonstrated in the lens ; in this process oxygen is not required. For the further catabolism of pyruvate, oxygen is sometimes used. There is a small amount of oxygen in the aqueous derived from the blood, but by which enzymes it is used in the lens is not yet clear. The essential process is probably anaerobic and in the lens there is present a number of enzymes of the type whereby pyruvate is broken down to lactic acid and water. As we have noted, lactic acid is found in considerable quantity in the aqueous humour when the lens is present ; this is not so in the aphakic eye. Agents which appear to participate in this process are glutathione and ascorbic acid (vitamin C) which, reacting together, probably participate in an internal autoxidative system. The former, both as reduced and oxidized glutathione, occurs in very high concentration in the lens, particularly in the cortex ; the latter we have seen to be specially secreted by the ciliary body. Neither is present in cataract. From the clinical point of view it is interesting, also, that the metabolic activity of the lens is largely confined to the cortex ; the older nucleus is relatively inert.

CHAPTER 3

THE PHYSIOLOGY OF VISION

WHEN light falls upon the retina it acts as a stimulus to the rods and cones which serve as the sensory nerve endings. As contact of the skin with a foreign substance causes the sensation of touch, so stimulation of the retina by light causes visual sensations ; upon these sensory cells the images of objects in the outside world are focused by the dioptric system of the eye. It follows that rays falling upon the optic disc give rise to no visual sensation and this is therefore called the *blind-spot* (of Mariotte). Light falling upon the retina, however, causes two essential reactions, photochemical and electrical.

The *photochemical changes* concern the pigments in the rods and cones. The most fully explored pigment is rhodopsin (visual purple), found in considerable quantity in the rods ; several related pigments have recently been discovered in the rods of various types of animal, while it would seem that three different pigments are associated with the foveal cones. Rhodopsin is a chromo-protein, the molecule of which consists of a reactive part, a chromophore, responsible for the preferential absorption of light, attached to a protein which acts essentially as a support. The chromophore belongs to the family of the carotenoids and when exposed to light it is broken down through several intermediaries to the colourless vitamin A, a reaction which is reversible. It is this photochemical reaction which initiates the visual process and gives rise to the changes in electrical potential which are transmitted through the bipolar cells to the ganglion cells and along the fibres of the optic nerve to the brain. The pigments in the cones have not yet been fully elucidated, but it is likely that each reacts preferentially to different bands of wavelength in the spectrum which are perceived as red, green and blue.

The study of the *electrical responses* which follow has yielded results of great interest although all their implications are not yet clear. When the retina is stimulated, electrical variations (action-potentials) occur in the optic nerve fibres, presumably initiated by the photochemical changes in the rods and cones. These are of the same type as occur in all sensory nerves ; they consist of biphasic variations always of the same amplitude (the all-or-none response) but varying in frequency with the intensity of the stimulation. In vertebrate eyes some fibres show a burst of activity at the onset of stimulation (the "on-effect"), others show activity while the stimulus lasts, and others again show a burst of activity, pre-

sumably inhibitory in nature, when stimulation ceases (the "off-effect"). Any single nerve fibre reacts when a considerable area of the retina is stimulated; this (the receptive field) varies in extent from a diameter of 0.5 to 1.0 mm. and indicates the synaptic link-up of each ganglion cell with a number of receptor cells. Moreover, differences in the reaction resulting from stimulation by isolated wavebands of light show that a neural mechanism exists capable of colour discrimination. Reaching the occipital cortex some 124 milliseconds after retinal stimulation, these impulses modify the electrical activity of the brain as recorded by the electro-encephalogram. A somewhat crude additive record of the electrical changes in the retina can be obtained clinically in the electroretinogram, a technique which can be of diagnostic value in retinal disease.

VISUAL PERCEPTIONS

We are more concerned, however, with the sensations which result from stimulation of the retina with light. These are of three kinds, which are called the Light Sense, the Form Sense, and the Colour Sense.

The Light Sense is the faculty which permits us to perceive light, not only as such, but in all its gradations of intensity. If the

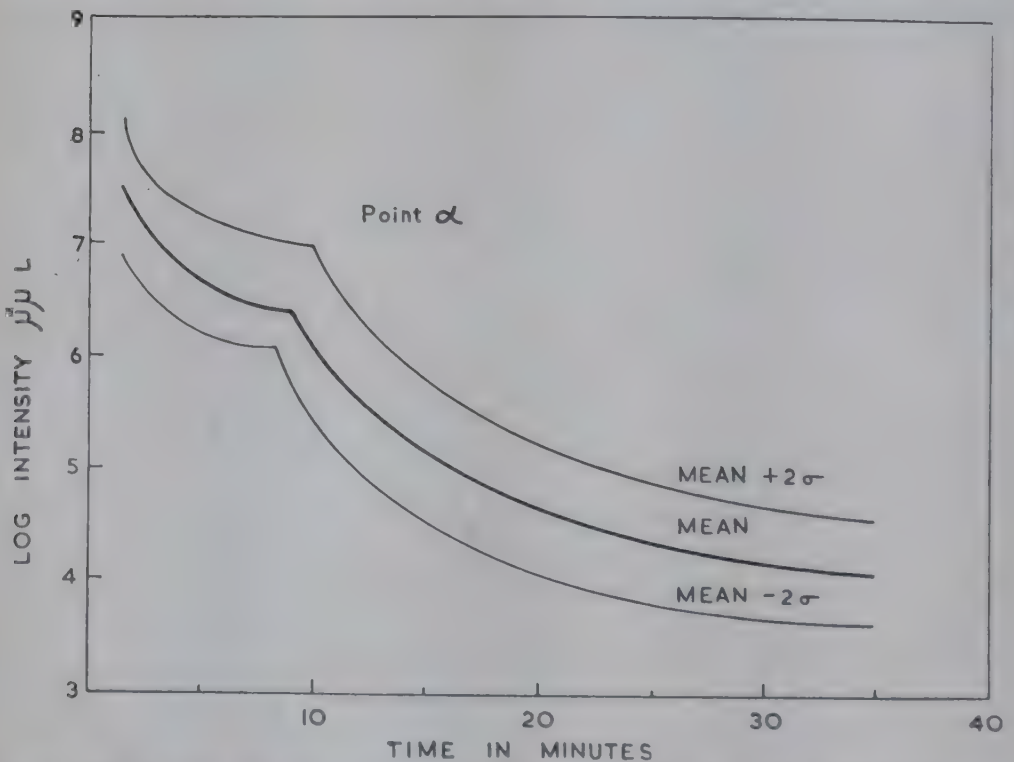


FIG. 25. Dark adaptation curve. The initial small symmetrical curve represents the adaptation of the cones. It is broken at a sharp knee (α) and the remainder of the curve represents the adaptation of the rods (after Sloan).

light which is falling upon the retina is gradually reduced in intensity there comes a point when it is no longer perceived : this is called the *light minimum*. It varies greatly according to the amount of light which has been falling upon the retina before the observation is made (*adaptation*). We are all aware that if we go from bright sunshine into a dimly lit room we cannot perceive the objects in the room until some time has elapsed : the eyes have to become " adapted " to the amount of illumination. Hence observations on the light minimum are only comparable when the eyes are in the same condition of dark adaptation as is obtained by excluding light from them for at least twenty to thirty minutes. The light minimum for the fovea is considerably higher than for the paracentral and peripheral parts of the retina, and retinal adaptation affects the macula relatively little (Fig. 25). It follows that in diseases which affect the rods particularly, much of the ability to adapt is lost and the patient is virtually night-blind.

The rods are much more sensitive to low illumination than the cones, so that in the dusk we see with our rods (*scotopic vision*) ; in bright illumination the cones come into play (*photopic vision*). Nocturnal animals, like the bat, have few or no cones ; diurnal animals, like the squirrel, have no rods ; man has an ample supply of both.

The Form Sense which is next in importance, is the faculty which enables us to perceive the shape of objects in the outer world. Here the cones play the predominant part, and the form sense is most acute at the fovea, where they are most closely set and

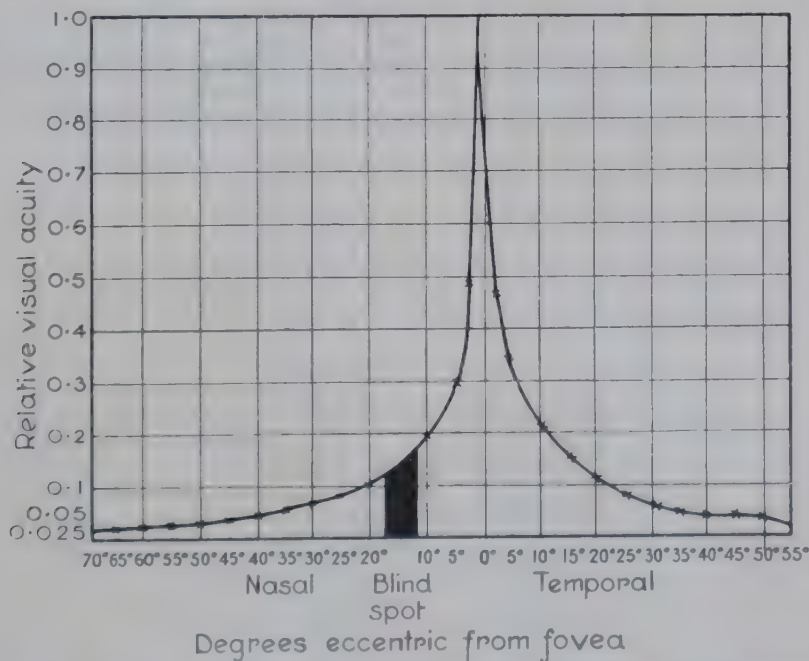


FIG. 26. The regional variations of the visual acuity in the retina.

most highly differentiated. It falls off very rapidly towards the periphery, as is shown in Fig. 26, and it is noticeable that the curve agrees fairly well with the diminution in the number of cones. We are accustomed to speak of the ability to distinguish the shapes of objects as the *visual acuity*, and we mean by that the greatest acuity which it is possible to obtain. The acuity of vision, therefore, applies to central vision, or the vision of objects the images of which are formed at the fovea.

It is to be noted, however, that the form sense is not a purely retinal function, for in the perception of composite forms—such as letters—it is largely psychological.

The Colour Sense is that faculty whereby we are enabled to distinguish between different colours as excited by light of different wavelengths. The appreciation of colours is a function of the cones and therefore occurs only in photopic vision, that is, with lights of moderate or high intensity and with some degree of light adaptation of the retina. In very low intensities of illumination the dark-adapted eye sees no colour and all objects are seen as grey, differing somewhat in brightness.

In different cones there are three pigments which absorb preferentially wavelengths of light in the spectrum corresponding to the colours red, green and blue, and if these (or, indeed, any three colours sufficiently far apart in the spectrum) are chosen, all the other colours as well as white light can be formed by their combination in suitable proportions. Hence normal colour vision is called *trichromatic*. This is the basis of the Young-Helmholtz theory of colour vision. In its original form this theory does not adequately explain all the phenomena associated with the appreciation of colour by normal individuals and in the colour-defective, but that there is a trichromatic stage in the visual process is undeniable.

CHAPTER 4

THE NEUROLOGY OF VISION

THE VISUAL PATHWAYS

IN the preceding chapter we have compared the process of vision with the results of the stimulation of other sensory nerves ; the comparison of the afferent tracts of common sensation with those of vision throws so much light upon the latter that it is worthy of a moment's consideration.

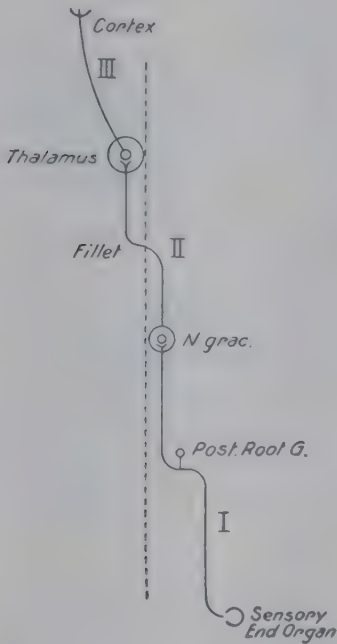


FIG. 27. The path of somæsthetic sensation.

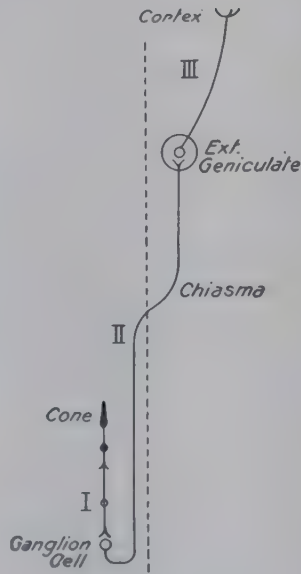


FIG. 28. The visual path.

The sensory impulse of common sensation in a limb is carried by a nerve fibre along the sensory nerve and the dorsal spinal root to the cord : it travels up in the posterior columns of the cord to the nucleus gracilis or the nucleus cuneatus as the case may be. The whole of this course is along the processes of a single cell or neurone, which has been called the neurone of the first order (I, Fig. 27). The impulse is taken up in the nucleus gracilis or cuneatus by a second cell, and is carried along the nucleo-thalamic tract or medial lemniscus to the opposite thalamus. The cells in the nuclei gracilis and cuneatus are the neurones of the second order (II, Fig. 27). A third cell, the neurone of the third order, situated in the thalamus, carries on the impulse to the cerebral cortex where the nervous impulse is transformed into a sensory perception, a change which is not and probably never can be understood.

Let us compare with this the visual afferent tracts (Fig. 28). The end-organ is the neural epithelium of rods and cones. The first conducting nerve cell or neurone of the first order is the bipolar cell of the inner nuclear layer of the retina with its axon in the inner reticular layer. This microscopic cell corresponds morphologically to a dorsal root ganglion cell and its long processes stretching, in some cases, from the tip of the toe to the top of the spinal cord. The neurones of the second order are the ganglion cells in the retina the processes of which pass into the nerve fibre layer and along the optic

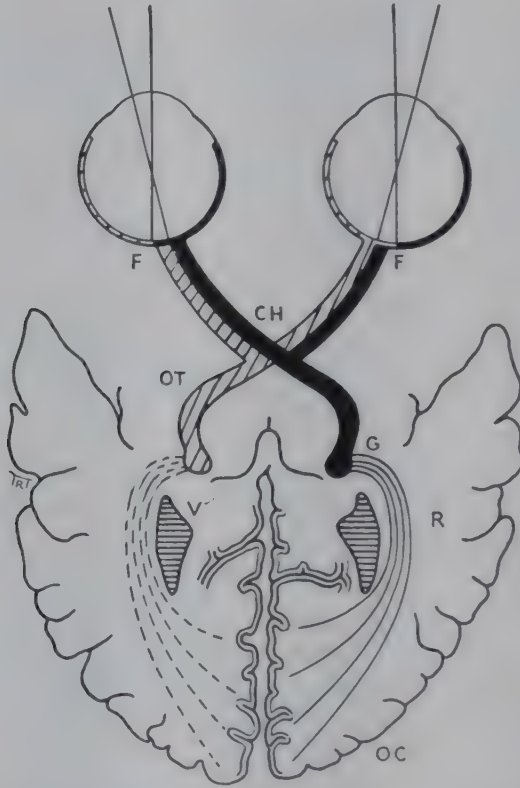


FIG. 29. The visual nerve paths showing lines of projection of the fixation area and the blind spot (from Traquair's "Clinical Perimetry"). F, fovea ; CH, chiasma ; OT, optic tract ; G, lateral geniculate body ; R, optic radiations ; OC, occipital cortex ; V, lateral ventricle.

nerve to the lateral geniculate body. Here a new cell, the neurone of the third order, takes up the transmission of the impulse, travelling by way of the optic radiations to the cortex of the occipital lobe, which is the so-called *visual centre*.

We see, then, the morphological identity of the two systems, in spite of the great anatomical differences which specialization has brought about. We may emphasize again the fact that the peripheral optic nerve proper is a bipolar cell in the inner nuclear and inner plexiform layers of the retina, while the so-called optic

nerve is a part of the central nervous system homologous with the medial lemniscus in the medulla and pons.

The course of the fibres from the various parts of the retina is seen in Fig. 29. In general it may be said that the fibres from peripheral parts enter the periphery of the optic nerve, while the fibres from parts of the retina near the optic disc enter the central parts of the nerve : they maintain this relative position as far back as the chiasma. The fibres from the macular region, however, form

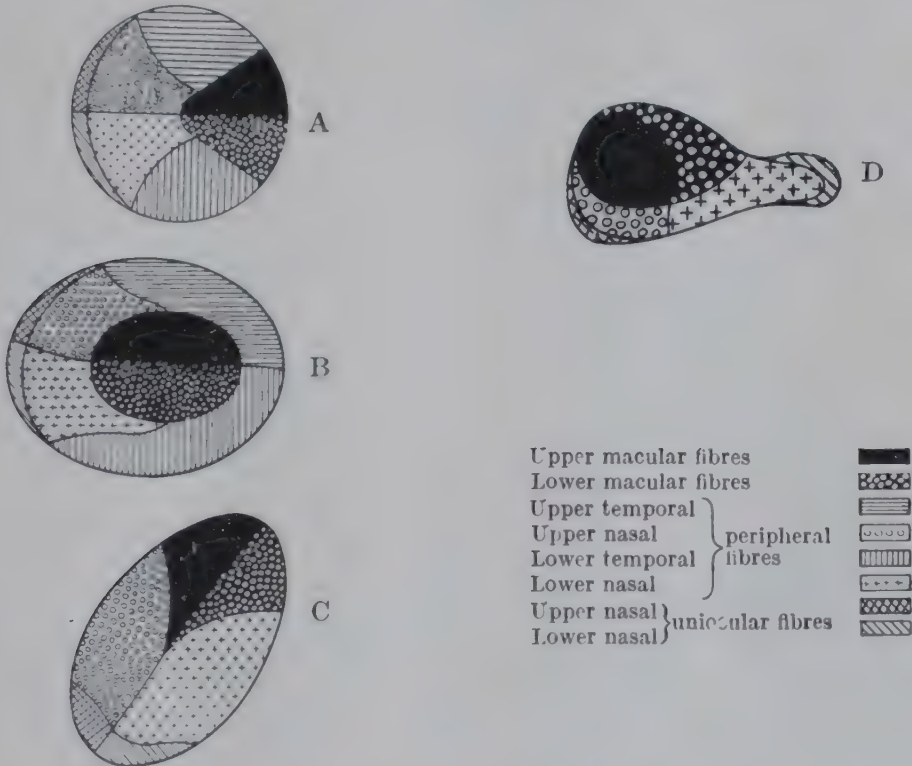


FIG. 30. The distribution of the fibres in the lower visual neurone of the right side. A, distal portion of the optic nerve; B, proximal portion of the optic nerve; C, optic tract; D, lateral geniculate body. In each case the dorsal aspect is above, the medial to the left.

a disturbing factor ; they enter the nerve on its outer aspect, where they are spread over an area which is triangular in section, with the apex towards the centre of the nerve (Fig. 30). These *papillo-macular fibres* soon become more centrally situated, so that in the posterior part of the nerve they are all in the centre. Tracing the nerve fibres still farther backwards, a partial decussation occurs wherein the nasal fibres cross in the chiasma, while the temporal ones enter the optic tract of the same side to reach the dorsal part of the lateral geniculate bodies. The axons of their corresponding neurones of the third order are also widely distributed in the central part of the optic radiations and end at the most posterior part of the visual

cortex at the tip of the occipital pole ; each half macula (R. and L.) is thus represented in the corresponding occipital pole (Fig. 29).

The fibres from peripheral regions of the retina similarly form two distinct groups, corresponding to the temporal and nasal halves of the retina. The distinction is very exact, as if a vertical line divided the retina into two halves at the level of the fovea (Fig. 29). The fibres from the temporal half of the retina enter the chiasma and pass into the optic tract of the same side ; thence they

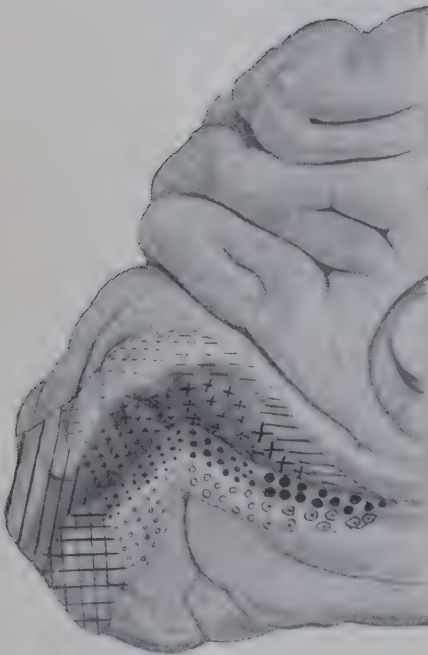


FIG. 31.

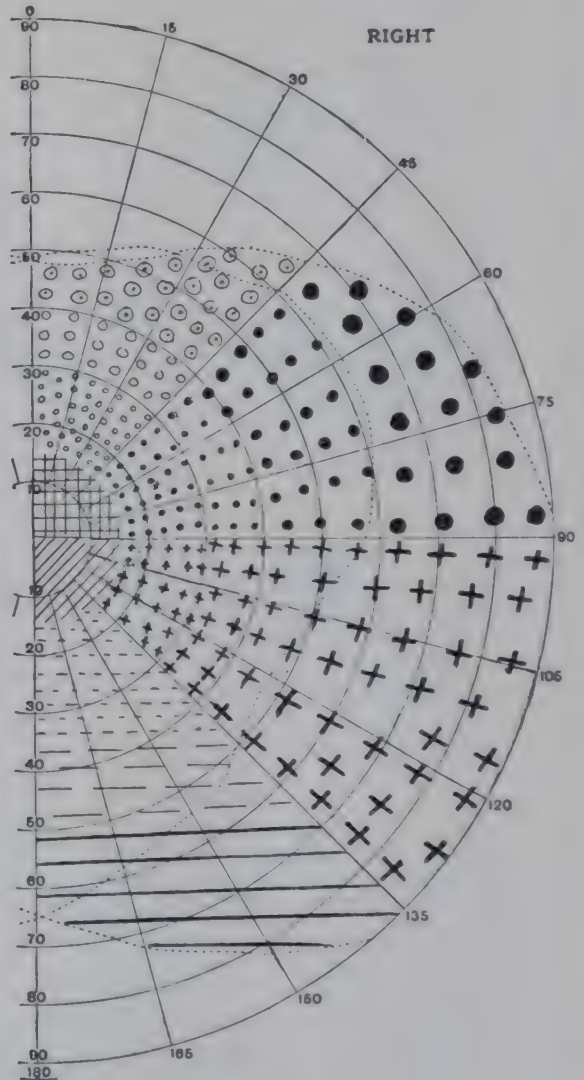


FIG. 32.

FIGS. 31-32. A diagram of the probable representation of the different portions of the visual fields in the calcarine cortex. Fig. 31 is a drawing of the medial surface of the left occipital lobe with the lips of the calcarine fissure separated so that its walls and floor are visible. The markings of the various portions of the visual cortex which is thus exposed correspond to those shown on the chart (Fig. 32) of the right half of the field of vision (Gordon Holmes).

run to the lateral geniculate body where all the visual fibres end. The fibres from the nasal half of each retina enter the chiasma, decussate, and pass into the optic tract of the opposite side, the arrangement being such that the direct and crossed fibres pass to alternating laminae in the lateral geniculate body. The corresponding neurones of the third order pass by the optic radiations to the corresponding occipital lobes. It follows that a lesion of one occipital lobe or optic tract will cause blindness of the temporal half of the retina on the same side and of the nasal half of the retina on the opposite side. Projecting this outwards, such a lesion will cause loss of vision in the opposite half of the binocular field of vision, a condition which is known as *hemianopia*. We shall see that the afferent pupillo-constrictor fibres have a similar semi-decussation in the chiasma (Fig. 35).

The visual fibres in the optic radiations, like other sensory tracts, run behind the motor fibres in the internal capsule. Thereafter they separate considerably, the ventral fibres (projecting the lower quadrant of the retina or the upper quadrant of the visual field) running forwards into the temporal lobe before they turn backwards to the lower portion of the visual cortex, the dorsal fibres (projecting the upper retinal quadrant or lower field) running backwards in a more direct course to the upper part of the visual cortex (Fig. 29). They pass close to the posterior cornu of the lateral ventricle, so that they are liable to pressure here when the ventricle is distended.

The occipital cortex in and about the calcarine fissure differs from the cortex elsewhere in the possession of a white line, the line of Gennari, interpolated in the grey matter. This area, which is the primary visual or visuo-sensory area (Figs. 31-32), is the cortical projection of the corresponding halves of both retinae. In this projection the same spatial arrangement is maintained—the part above the calcarine fissure represents the upper corresponding quadrants, the part below represents the lower corresponding quadrants of both retinae, and the posterior part of the occipital lobe represents the macula.

THE PUPILLARY PATHWAYS AND REACTIONS

We have seen that the pupils are controlled by two muscles of ectodermal origin like the *erectores pili* of the skin—the sphincter and dilatator. The responses of these muscles to stimuli are very rapid and delicate and are easily observed, and the size of the pupil may be looked upon as essentially the resultant of their opposing forces. The constrictor centre possesses “tone” and is perpetually sending out impulses to the sphincter which keep the pupil slightly contracted. Abnormal enlargement of the pupil is called *mydriasis*, abnormal contraction, *miosis*.

When the responses are altered by disease, the changes which occur afford valuable information about the condition of the nerve tracts involved. These tracts are somewhat complicated, but it is essential that they should be understood.

The Pupillary Pathways. The innervation of these muscles is seen in Figs. 33 and 34.

The sphincter is supplied by cholinergic nerves of the parasympathetic system through the third cranial nerve. The fibres start in the Edinger-Westphal nucleus near the third nucleus in the floor of

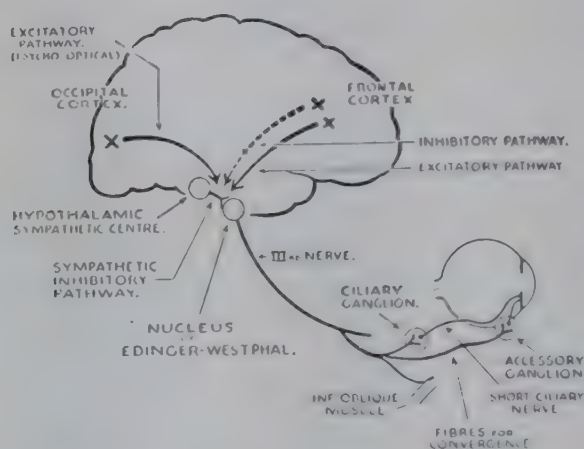


FIG. 33. The parasympathetic pupillary system.

Cortical control: (a) excitatory pathways from the frontal and occipital (psycho-optical) cortex ; (b) inhibitory pathway from the frontal cortex.

Sympathetic control (inhibitory) from hypothalamic centre.

Pathway—Edinger-Westphal nucleus → third nerve → inferior division → branch to inferior oblique →

(a) *Light reflex*—short root of ciliary ganglion → ciliary ganglion → short ciliary nerves → sphincter of iris.

(b) *Near reflex*—leaving the third nerve at an unknown point → (?) accessory ganglion → sphincter of iris.

the aqueduct of Sylvius. This nucleus has connections with the dilatator centre as well as with the frontal and occipital cortex. From it the fibres pass out of the mid-brain and run in the main trunk of the third nerve as far as the orbit. Here the fibres pass into the branch which supplies the inferior oblique muscle, leaving it by the short root of the ciliary ganglion. From the ciliary ganglion they pass by the short ciliary nerves to the eye, piercing the sclera around the optic nerve in company with the short ciliary arteries. The nerve fibres pass forwards in the choroid and ciliary body to the iris.

The dilatator pupillæ is supplied by the adrenergic fibres of the cervical sympathetic nerve (Fig. 34). The dilatator tract probably commences in the hypothalamus not far from the constrictor centre, and it also has connections with the cerebral cortex.

From the hypothalamic centre the dilatator fibres pass downwards through the medulla oblongata into the lateral columns of the cord. The fibres leave the cord by the ventral roots of the first three dorsal and probably the last two cervical nerves, enter the rami communi-

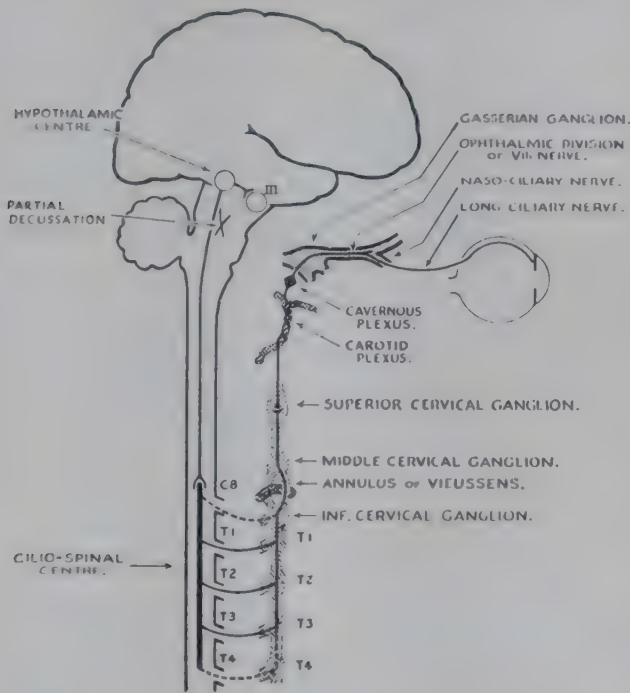


FIG. 34. Sympathetic pupillary system.

I. CENTRAL. Centre in hypothalamic region—inhibitory pathway to Edinger-Westphal group of Nucleus III. *Tract* (hypothalamic-spinal) with a partial decussation in the mid-brain so that each hypothalamic centre supplies each cilio-spinal centre but especially the contralateral: it traverses the reticular substance of the mid-brain and the lateral column of the cord.

II. PRE-GANGLIONIC SYMPATHETIC. Cilio-spinal centre of Budge in the intermedio-lateral tract of the grey matter of the cord. Leaves by ventral roots of C 8, T 1, 2 and 3 (largely T 1) *via* the white rami communicantes → cervical sympathetic chain in which the fibres traverse the inferior cervical ganglion and the anterior loop of the ansa of Vieussens and terminate in the superior cervical ganglion.

III. POST-GANGLIONIC SYMPATHETIC. Sup. cerv. gang. → enter the skull with the carotid plexus → cavernous plexus → travel over the semilunar ganglion → along first division of V → naso-ciliary nerve → long ciliary nerves, entering the globe with the long ciliary arteries (some perhaps running without a relay along the long and/or sympathetic root of the ciliary ganglion → short ciliary nerves), traversing the epichoroidal space to reach the iris and terminate in the dilatator muscle.

cantes, and run to the first thoracic or stellate ganglion. From here they pass by the anterior limb of the ansa of Vieussens into the cervical sympathetic. In this nerve they run up the neck to the superior cervical ganglion, whence they pass with the carotid plexus into the skull. They run over the anterior part of the semilunar ganglion and pass into the first or ophthalmic division of the fifth nerve, following

the nasal branch, which they finally leave to enter the long ciliary nerves, thus avoiding the ciliary ganglion. The long ciliary nerves enter the eye on each side of the optic nerve, accompanying the long ciliary arteries. Like them, they run forwards between the choroid and sclera, enter the ciliary body and thus reach the iris.

The balance of tone between these two antagonistic innervations maintains the pupil at its normal size, the essential factor being the superior tone of the sphincter. The pupils are normally equal on the two sides ; it is rare to meet with unequal pupils (*anisocoria*) in a normal person ; such cases do occur, but every pathological cause must be eliminated before we rest content that the condition is an idiosyncrasy. On the other hand, the size of the pupils varies much in different people under the same conditions of illumination. In old people it is smaller than in the young, sometimes to so great an extent that the pupils are almost "pin-point." They are often smaller in hypermetropes, and larger in myopes than in emmetropes and are commonly smaller in blue eyes than in brown.

The Pupillary Reflexes. The pupils participate in several reflexes, three of which are of clinical importance :

1. The *light reflexes*, whereby if light enters an eye the pupil of this eye contracts (the *direct light reflex*), an activity shared equally by the pupil of the other eye (the *consensual light reflex*).

2. The *near reflex*, whereby a contraction occurs on looking at a near object, a reflex largely determined by the reaction to convergence, but in which accommodation also plays a part.

3. The *psycho-sensory reflex*, whereby a dilatation occurs on psychic and sensory stimuli.

The *light reflex* is initiated from the rods and cones throughout the retina. The fibres run up the optic nerve, partially decussate in the chiasma and enter the optic tracts with exactly the same distribution as the visual fibres (Fig. 35). Near the upper end of the tract, however, they part company with these and, instead of running to the lateral geniculate body, they enter the pretectal region. Here they are relayed in a small *pretectal nucleus*, and the new fibres, suffering a partial decussation in the mid-brain, travel to the Edinger-Westphal nucleus on each side. From these nuclei the constrictor fibres travel to each iris as already described.

The decussation is important for it explains the mechanism of the consensual as well as the direct reaction to light and also accounts for several pathological reactions such as the Argyll Robertson pupil (*q.v.*). It is obvious from a study of these paths that a lesion distal to the chiasma will abolish the direct reaction in the eye on the affected side and the consensual reaction on the other (Fig. 35) ; a lesion in the optic tract will produce a hemianopic reaction involving both eyes, while blindness due to a lesion affecting the visual pathways in or above the lateral geniculate body will leave the

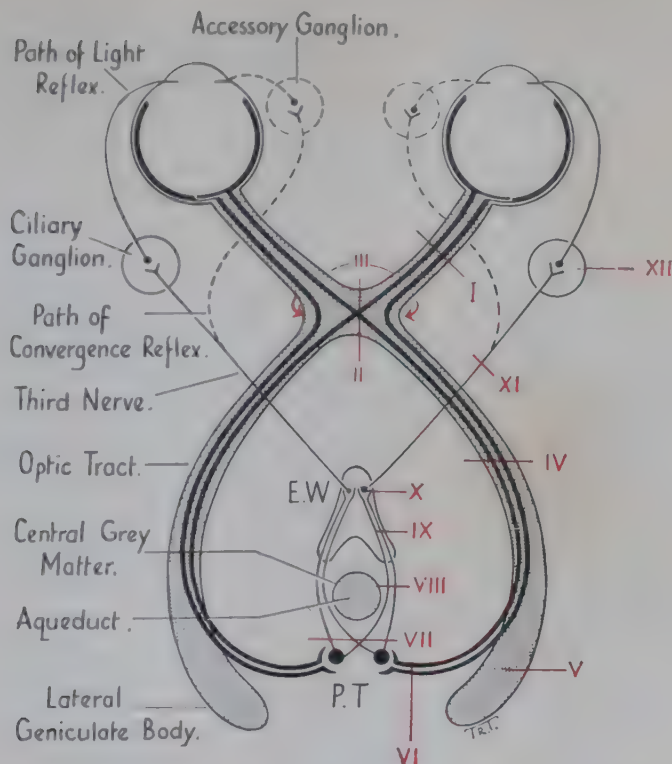


FIG. 35. The pupillary pathways for the light reflex. E.W., Edinger-Westphal nucleus; P.T., pretectal nucleus. The numbers in red denote lesions accompanied by the following symptoms:—

I. Optic nerve: *unilateral amaurotic paralysis* (abolition of the direct reaction on the ipsilateral side and the consensual on the contralateral side; retention of the consensual on the ipsilateral side and the direct on the contralateral side). Retention of the near reflex and the lid reflex.

II. Medial chiasma: *bitemporal hemianopic paralysis*.

III. Lateral chiasma: *binasal hemianopic paralysis*.

IV. Optic tract: *contralateral hemianopic paralysis* (Wernicke's reaction).

V. Lesion of the proximal part of optic tract: (normal pupillary reactions).

VI. Superficially in the region of the brachium and tectum: *contralateral hemianopic paralysis*.

VII. Central decussation: *bilateral reflex paralysis*—inactivity to light (direct and consensual) with retention of the near reflex, the lid reflexes and the psycho-sensory reactions (*bilateral Argyll Robertson pupil*) (according to Behr).

VIII. Between the decussation and the constrictor centre: *ipsilateral abolition of direct and consensual reactions with retention of both contralaterally—unilateral Argyll Robertson pupil* (according to Behr).

IX. A partial lesion corresponding to VIII: *ipsilateral abolition of direct reaction with retention of consensual reaction; retention of both contralaterally*.

X. Nuclear or extensive supranuclear lesion: *ipsilateral absolute pupillary paralysis*.

XI. Lesion of IIIrd nerve: *absolute pupillary paralysis*.

XII. Lesion of ciliary ganglion: *abolition of the light reflex with retention of the near reflex (Argyll Robertson pupil)*.

pupillary reactions unaltered. It is also obvious that if one eye sees and the other is blind, stimulation of the first by light will elicit the consensual reaction in the second, provided the reflex

pathways in the mid-brain and third nerve are intact and the iris of this eye functioning.

The *near reflex* is initiated mainly by fibres from the medial rectus muscles which contract on convergence (Fig. 36). From these muscles afferent fibres run centrally, probably by the third nerve to the mesencephalic nucleus of the fifth nerve, to a presumptive convergence centre in the tectal or pre-tectal region. From this the pathway is relayed to the Edinger-Westphal nucleus and along the third nerve to the sphincter muscle of the iris, so that the pupil contracts commensurately with convergence. At the same time, accommodation reinforces the reflex by visual impulses relayed from the cortex to the Edinger-Westphal nucleus.

The *sensory reflex*, which is initiated by the stimulation of any sensory nerve to the extent of causing pain or by emotional states and excitement, is more complicated than the light reflex, for both the dilatator and the constrictor centres play a part in its production. Sensory stimulation causes first a rapid dilatation of the pupil due to augmentation of the dilatator tone through the cervical sympathetic, and then a second dilatation, rapid in onset but slow in disappearance, due to inhibition of the constrictor tone.

Minute examination of the pupil when the intensity of the light entering the eye is altered, shows that the pupil contracts and then oscillates rapidly, finally settling down into a condition of contrac-

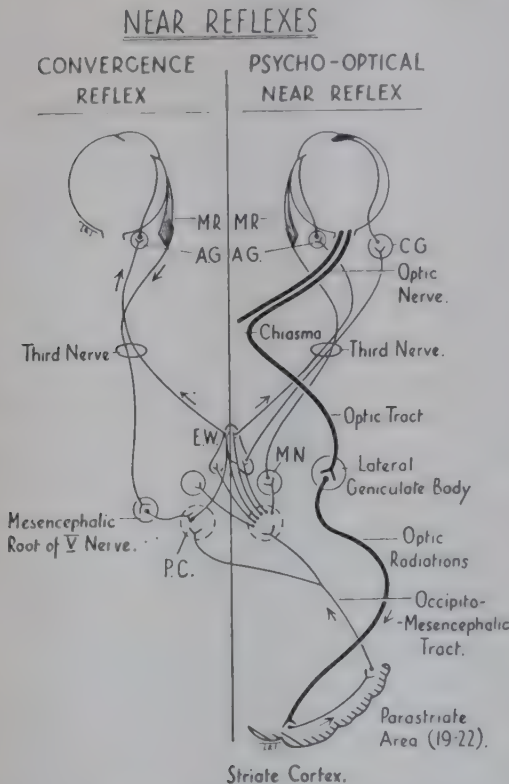


FIG. 36. The nerve paths of the two components of the near reflex. The afferent pathway for the convergence reflex is indicated as running up the IIIrd nerve: this is not certain. That for the accommodation reflex follows the visual fibres to the striate area of the calcarine cortex (area 17), is relayed to the parastriate area (19), whence the efferent path travels to the Edinger-Westphal nucleus *via* the occipito-mesencephalic tract and the pontine centre for convergence. A.G., accessory ganglion; C.G., ciliary ganglion; E.W., Edinger-Westphal nucleus; M.N., nucleus for medial rectus; M.R., medial rectus; P.C., pontine centre.

tion which is slightly less than the summit of the first wave. In its sudden response, the pupil as it were oversteps the mark, oversteps it again in the opposite direction, and so on. Two different types of exaggeration of this oscillation are met with in abnormal conditions. One is the condition in which the oscillations are very large and easily seen, and are to a great extent independent of the light falling upon the eye. This is called *hippus*; it depends upon the rhythmic activity of the nervous centres, and is not a peripheral phenomenon. More important is the lack of sustained contraction under the continued influence of light. Here the pupil contracts sluggishly when the intensity of the light is increased, but while the light is still kept constant it slowly dilates, often with superimposed sluggish oscillations. This is a pathological phenomenon dependent upon diminished conductivity in the afferent path of the light reflex, usually in the optic nerve as in optic neuritis.

The Action of Drugs on the Intra-ocular Musculature.

Drugs are so frequently employed in ophthalmic practice for the purpose of dilating or constricting the pupils or paralysing the accommodation, that it is important to know exactly how they act. Pupil-dilating drugs are called *mydriatics*; pupil-constricting, *miotics*; drugs which paralyse the ciliary muscle, *cycloplegics*. All drugs which dilate the pupil also paralyse the accommodation in greater or less degree; similarly, all miotics stimulate the ciliary muscle to contract, so that the eye assumes a condition of partial or complete accommodation.

All these drugs, when instilled into the conjunctival sac, are rapidly absorbed through the cornea and become effective in the inner eye. Constriction of the pupil by the third nerve is due to the liberation of acetylcholine which, almost as soon as it has been formed and has accomplished its immediate purpose, is destroyed by an enzyme, cholinesterase. One type of drug, of which atropine is the prototype, abolishes the action of acetylcholine and thus causes mydriasis by making it impossible for the sphincter to contract (*parasympatholytic drugs*). A second type of drug acts as a parasympathetic stimulant and thus causes miosis (*cholinergic drugs*); this can be done in two ways. (1) The drug may act as a direct stimulant on the myoneural junction, supplementing the normal effect of acetylcholine (the acetylcholine-like drugs, of which pilocarpine is the prototype). (2) The drug may act as an indirect stimulant by abolishing the effect of cholinesterase so that the acetylcholine formed on the activity of the third nerve continues its effect and a sustained miosis results (*anticholinesterase drugs*, of which eserine is the prototype).

A further class of drugs acts on the sympathetic which, it will be remembered, exerts its activity at the myoneural junctions owing to the liberation of noradrenaline; when it has performed its func-

tion, noradrenaline (like acetylcholine) is destroyed by oxidative enzymes. As with the parasympathetic mechanism, drugs are available which stimulate the sympathetic apparatus either by stimulating the myoneural function directly or by suppressing the action of oxidases. The prototype of such *sympathomimetic drugs* is adrenaline which produces a mydriasis. The opposite effect of inducing a miosis by a paralysis of the sympathetic (*sympatholytic drugs*) is less commonly used in ophthalmology.

Finally, a miosis may be induced by direct stimulation of the muscle cells by a drug such as histamine.

Parasympatholytic mydriatic drugs. *Atropine* is the strongest mydriatic at our disposal ; it completely paralyses the sphincter pupillae and ciliary muscle. It takes a considerable time to cause complete paralysis ; one drop of 1·0 per cent. atropine sulphate solution causes wide dilatation of the pupil in thirty to forty minutes, and marked paralysis of accommodation in about two hours ; the effects do not pass off entirely for three to seven days.

Homatropine acts more quickly than atropine, and the effects pass off rapidly ; as a 1·0 per cent. solution its effect is fully evident in three-quarters of an hour, especially if combined with cocaine (2 per cent.) which facilitates absorption and acts synergically by stimulating the dilatator mechanism. The effects pass off completely in forty-eight hours, or much more quickly if a drop of eserine (0·5 per cent.) is instilled. The mixture of homatropine and cocaine which is commonly employed for estimating refraction does not abolish the tone of the ciliary muscle so fully as atropine.

Scopolamine (hyoscine) (0·1 to 1·0 per cent.), and *duboisine (hyoscyamine) sulphate* (0·5 per cent.) have a similar but less powerful action than atropine.

Other useful drugs of this type which have been recently introduced are *cyclopentolate hydrochloride* (Cyclogyl, Mydrilate) (0·5 to 2 per cent.) and *bistropamide* (Mydriacyl) the reactions of which are both rapid and temporary.

Sympathomimetic mydriatic drugs. *Adrenaline (epinephrine)* which acts on the dilatator fibres directly, produces dilatation after the instillation of four drops of a 1 in 1,000 solution, the instillation being repeated in five minutes. The effect is evident in half an hour.

Cocaine, besides its anæsthetic effect upon the endings of the fifth nerve, also stimulates the sympathetic nerve endings in the dilatator pupillae. It does not paralyse the sphincter, so that the dilatation of the pupil is only moderate, and the pupil continues to react to light even after prolonged application. Cocaine is therefore ineffective and adrenaline effective when the sympathetic nerve is paralysed.

Other drugs in this class are *phenylephrine (neo-synephrine hydrochloride)*, *ephedrine*, *paredrine (hydroxyamphetamine)* and *benzedrine (amphetamine)*.

Parasympathetic stimulatory miotics. (a) Direct stimulants (acetylcholine-like drugs).

Pilocarpine (0·25 to 5·0 per cent.) causes miosis by directly stimulating the myoneural junctions of the sphincter muscle, and is thus still active after degeneration of the nerve fibres. The action is not prolonged and may be followed by a fatigue-reaction—slight mydriasis.

Other drugs in this class are *metacholine chloride (mécholyl chloride)* (10 to 20 per cent.), usually used with neostigmine, and *furmethide (furfuryl-trimethyl-ammonium-iodide)* (10 per cent.).

(b) Indirect stimulants (anticholinesterase drugs). *Eserine (physostigmine)* (0·25 to 1·0 per cent.) is a powerful and useful miotic. It is able to overcome the dilatation produced by 1 per cent. atropine only with difficulty. On the other hand, it readily overcomes the dilatation produced by homatropine and

cocaine. Owing to its mode of action eserine fails to produce constriction of the pupil after blocking of the third nerve. Normally it begins to contract the pupil and cause spasm of accommodation in about five minutes ; its maximum effect is reached in twenty to forty-five minutes. The effect on accommodation lasts only an hour or two, that on the pupil two to three days. When instilled into the conjunctival sac it causes some smarting and an unpleasant " dragging " sensation ; indeed, it may be so irritating as to cause vomiting, but this only occurs in very sensitive persons or when the drug is pushed. Owing to these symptoms it should not be instilled more frequently nor in stronger doses than are necessary to ensure the desired result. A 0·5 per cent. solution or one considerably weaker is often adequate.

Other drugs in this class are *neostigmine bromide* (*Prostigmin*) (3 to 5 per cent.), less powerful than eserine, *demecarium bromide* (*Humorsol*, *Tosmilen*) (0·5 per cent.), and three very powerful organophosphates, *di-iso-propyl-phosphorofluoridate* (*DFP*) (0·1 per cent.), *tetra-ethyl-pyrophosphate* (*TEPP*) (0·1 per cent.), and *echothiophate iodide* (*Phospholine*) (0·06 to 0·25 per cent.).

Two drugs are sometimes employed which combine the direct and indirect methods of stimulation—*doryl* (*carbachol*) (*carbamyl-choline chloride*) (1·0 per cent.) and *urecholine* (*carbaminoyl- β -methyl-choline chloride*) (1·0 per cent.). Both of these require the assistance of a wetting agent (*zephiran chloride*) (p. 139) to traverse the cornea with ease.

Sympatholytic miotics such as *ergotamine*, *ergotoxin*, *Priscol*, and *dibenamine* are effective on systemic administration but are rarely used clinically.

Histamine (" amino-glaucosan ") produces maximum miosis by acting directly upon the sphincter muscle fibres.

The action of these drugs may be used to determine whether an anomaly of the size of the pupil is due to an irritative or a paralytic lesion of either of the two nerves.

SECTION II

OPHTHALMIC OPTICS

CHAPTER 5

ELEMENTARY OPTICS

It is obvious that sharp images of external objects must be formed upon the retina if these are to be clearly seen. Before considering how this is effected it will be advisable to outline the elementary principles of optics. The student should realize that success in the diagnosis, and hence in the treatment, of diseases of the eye is impossible if such elementary principles of optics as are set forth here are not thoroughly mastered.

If white light, such as sunlight, is passed through a suitable prism or diffraction grating a spectrum is formed, consisting of rays differing from each other in wavelength. Of these certain are visible and appear to the majority of people as pure colours—red, orange, yellow, green, blue, and violet in the order named, the red having the longest and the violet the shortest wavelength. The visible spectrum extends from about $723\text{ m}\mu^*$ at the red end to $397\text{ m}\mu$ at the violet end, or roughly from $700\text{ m}\mu$ to $400\text{ m}\mu$. Beyond the red end are infra-red rays of greater length which, when absorbed, cause a rise in temperature and are commonly known as heat rays. Beyond the violet end are waves of smaller length, the ultra-violet rays, which are capable of causing chemical actions. The longer visible rays also cause a rise in temperature, and the visible rays are also actinic, though less so than the infra-red and ultra-violet respectively. Glass absorbs some of the heat rays and many of the ultra-violet but prisms and lenses made of quartz allow most of the ultra-violet rays to pass unimpeded. The media of the eye are uniformly permeable to the visible rays between $660\text{ m}\mu$ and $390\text{ m}\mu$ but the cornea absorbs rays shorter than $295\text{ m}\mu$, the lens rays shorter than $350\text{ m}\mu$, and the vitreous has an absorption band with its maximum at $270\text{ m}\mu$. Rays between $400\text{ m}\mu$ and $295\text{ m}\mu$ can therefore reach the lens, those between $400\text{ m}\mu$ and $350\text{ m}\mu$ can reach the retina in the normal eye, and those between $400\text{ m}\mu$ and $295\text{ m}\mu$ can reach the retina in an eye from which the lens has been removed. Whenever absorption occurs there is the possibility of pathological changes resulting. Sunlight at sea-level is poor in ultra-violet rays, which are absorbed in the atmosphere. Ordinary glass used for spectacles absorbs

* A $\text{m}\mu$ is a convenient unit of length equal to 1 millimicromillimeter, or 10^{-6} mm. ; in the terminology standardized in 1960, the term nanometer (nm.) has the same significance.

rays beyond 350 $m\mu$. Heat radiation from 1,100 $m\mu$ to 700 $m\mu$ passes into the eye almost unchecked, and a large amount of it reaches the retina. The pigment epithelium on the back of the iris absorbs radiation of all wavelengths, and the same is true of the retinal pigmentary epithelium at the back of the eye.

RAYS OF LIGHT AND IMAGES

It is a familiar fact that a naked light emits light in all directions. This is transmitted in straight lines, so that we may imagine it coming from the source as an immense number of diverging straight lines, each of which is called a *ray*. Every point on such a ray represents, or is the image of, the point of light from which it springs.

This is shown by a simple experiment carried out in a dark room. Make a pinhole in a piece of cardboard (A, Fig. 37) and hold the cardboard in front of the candle (C) at a little distance from it. Beyond the cardboard hold up a white screen (B), so that the cardboard is between the screen and the candle. A dim image (D) of the flame will be thrown upon the screen, and it will be noticed that it is upside down so that an inverted image of the flame is formed. This is due to the fact that the cardboard cuts off all the rays of light from the candle except such as can pass through the hole. The only rays from the top of the flame which can pass through the hole are those which are caught upon the lower part of the screen. The image is dim because only a few rays of light can pass through the small hole. Now make another hole a little distance away from the first. Another inverted image of the flame is seen. If a dozen holes are made, a dozen images appear and if the holes are close together the images will overlap. If a large hole is made, so many images overlap that all resemblance to the original flame is lost, and part of the screen becomes uniformly illuminated. If we take away the cardboard altogether the whole screen becomes illuminated, and we now know that this is because we have an infinite number of images of the flame all overlapping each other.

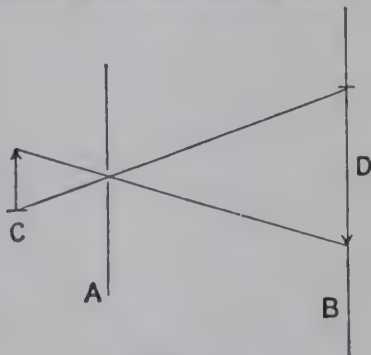


FIG. 37.

The speed of light varies when it traverses different substances. If the velocity is less in one medium than another, the first medium is said to be optically denser than the second.

When light, travelling in one medium, meets another medium it breaks up into two parts : part is *reflected* back into the first medium ; part is *refracted* into the second medium. If the second medium is opaque none of the light is refracted.

REFLECTION

Let us now consider what happens to a ray of light when, travelling in one medium, it is reflected from the surface of a denser medium. Before it meets the surface it is called an *incident ray* ; after it is reflected from the surface it is called the *reflected ray*. If a line is drawn at right angles to the surface at the point where the incident ray meets it (the *normal*), it is found to be an invariable rule

that the incident ray makes the same angle with this line as the reflected ray. Put in formal language, this law of reflection is that *for all surfaces the angle of incidence is equal to the angle of reflection, and is in the same plane with it* (Fig. 38).

These principles can now be applied to the types of mirror which interest the ophthalmologist.

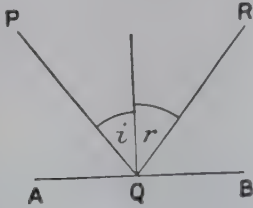


FIG. 38. The ray from P which strikes the mirror AB at Q is reflected to R so that PQ and QR are in the same plane, i.e., that of the paper, and the angle of incidence, i , is equal to the angle of reflection, r .

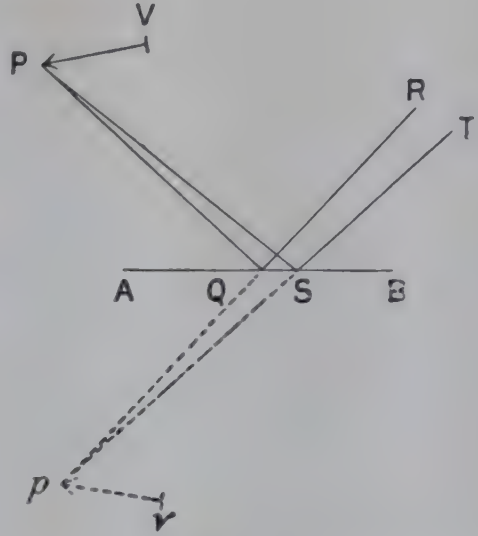


FIG. 39.

Plane Mirrors. If P (Fig. 39) is a luminous point in front of the mirror AB, the ray PQ will be reflected towards R, and the ray PS towards T; thus the reflected rays QR and ST appear to come from p , a point as far behind the mirror as P is in front of it. As the rays QR and ST have to be produced backwards in order that they may meet, no real image is formed, and such an image is called a *virtual image*. Note that the rays reflected from a plane mirror are divergent. The same reasoning holds good for every point on the object PV, its image being p_v , as far behind the mirror as the object is in front of it: moreover, the size of the image is equal to that of the object.

Concave Mirrors. Here the normal to the surface is the radius of the sphere of which the mirror forms a part; around any radius an incident ray must be symmetrically reflected. If AH (Fig. 40) is part of the section of a concave mirror and K is the centre of the sphere, then the line HKB is called the *axis* and H the apex of the mirror. The ray PK through the centre of the sphere will obviously be reflected along itself, so that the image

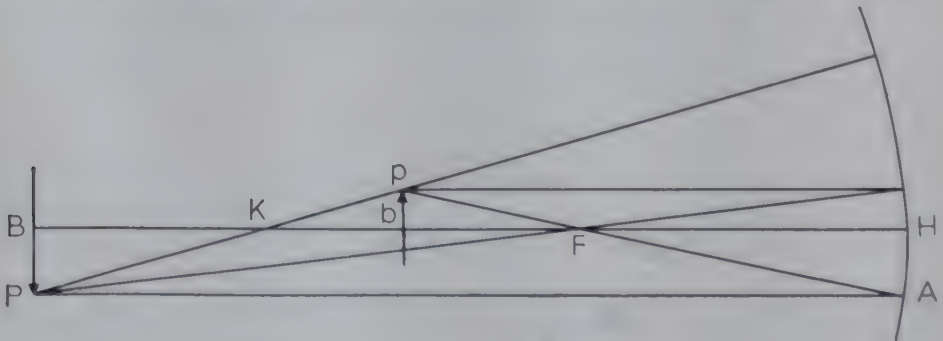


FIG. 40.

of P must be on PK . The ray PA , parallel to the axis and reflected symmetrically around the radius, will meet PK in p . Hence p is the image of P . Now it is found that all rays parallel to the axis and not very far removed from it cut the axis in the same point, F , and this point bisects the line HK . This point is called the *principal focus* of the mirror. If the object PB were removed a very great distance away from the mirror, all the rays which fell upon a small portion of the mirror near H would diverge so little from each other that they would all be practically parallel to BH , and the image of PB would be extremely small and situated at F . In each of these cases the image is an inverted one of the object.

It is an axiom of optics that the direction of the rays is reversible. Hence, if pb were an object, it would have its image at PB , and if there were an object at F , all the rays from it reflected by the mirror would be parallel to the axis, and the image would be infinitely large and situated at infinity.

If the object were situated between F and H (Fig. 41), the rays would diverge

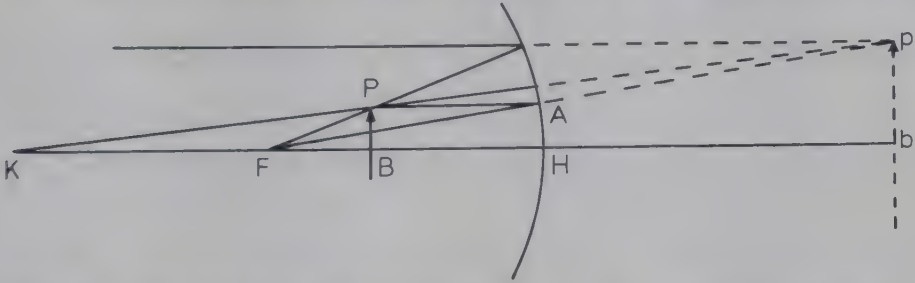


FIG. 41. The ray from P parallel to the axis is reflected through F , the principal focus. The ray FP is reflected parallel to the axis. The ray KP is normal to the surface, and is therefore reflected on itself. The meeting point of any two of these rays will give the situation of p , the image of P .

after reflection as if they came from an object behind the mirror, much as they do with a plane mirror. The image would therefore be a virtual one, situated behind the mirror: it would be erect and larger than the object.

The important fact to remember with regard to concave mirrors is that if the object is farther away from the mirror than its focal distance, i.e., than half its radius of curvature, the image is real and inverted, situated also in front of the mirror. This is the condition which is almost always present in the ordinary use of ophthalmic instruments.

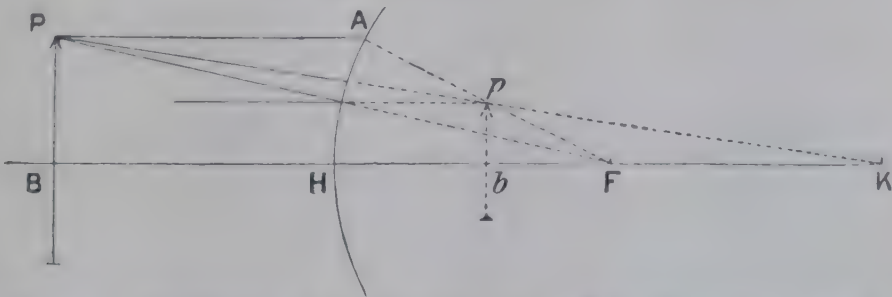


FIG. 42. Reflection by a convex mirror. The description of Fig. 41 applies equally to this figure.

Convex Mirrors. We are not accustomed to using convex mirrors in ophthalmic instruments, but it is necessary to know what happens with them, since the cornea acts as a convex mirror. By the same construction, as will be seen from Fig. 42, the image is always virtual, erect, and smaller than the object.

As with the concave mirror, if the object is a long way off, the image will be situated at the principal focus, i.e., at a distance equal to half the radius of curvature behind the mirror.

REFRACTION

We have now to consider what happens to the refracted ray when the incident ray, travelling in one medium, e.g., air, meets an optically denser medium, e.g., glass. Since the light will now travel more slowly it will be deviated towards the normal to the surface, and it will be more deviated the greater the difference in optical density between the two media. If the density of air is taken as unity, then the ratio of its density to that of the second medium is called the *index of refraction* of the medium.

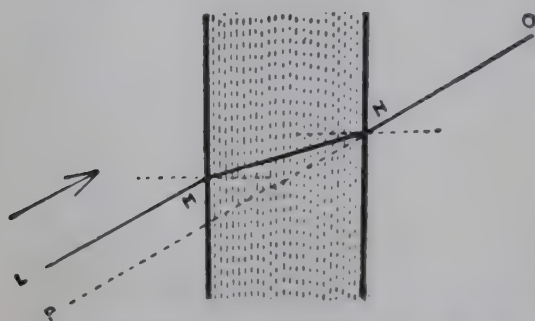


FIG. 43. Refraction by a plane lamina.

Plane Lamina. When an incident ray, such as LM (Fig. 43), meets the surface of a plate of glass with parallel sides it will be deflected towards the normal at M. When the ray passes out of the glass on the other side, it will obviously be deflected away from the new normal at N just as much as LM was deflected towards it. Hence the emergent ray NO will be parallel to the incident ray LM. If the plate of glass is very thin, NO will be practically continuous with LM.

Prisms. If we imagine the two sides of the plane lamina to meet at a point A, a prism will be formed (Fig. 44). In this case, being similarly refracted with reference to the normals at these surfaces, the ray will be deflected along DEFG. The ray is thus deviated towards the base of the prism. When the angles of incidence and emergence are equal, the ray is said to pass symmetrically through the prism. In these circumstances, if the prism is made of crown glass, the angle of deviation of the ray (DKH) is approximately equal to half the refracting angle of the prism.

We are accustomed to project objects along the direction of the rays of light as they enter the eye, and in doing so we ignore the effect of refraction, since it enters relatively little into our everyday experience. If, therefore, we look at a light D through a prism, as in Fig. 44, the light

will appear to come from H. Objects, then, seen through a prism, appear displaced towards the apex of the prism. Prisms may be

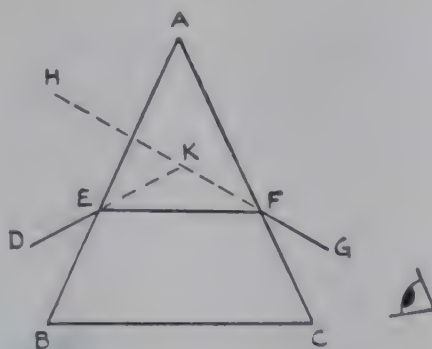


FIG. 44. Refraction by a prism.

categorized according to the apical angle or the angle of deviation, but more usually in *prism dioptres* (Δ), a unit indicating the strength of the prism which will produce a linear apparent displacement of 1 cm. of an object situated 1 metre away.

Lenses. Ordinary lenses are pieces of glass with spherical surfaces. The line passing through the centres of curvature of the surfaces is called the axis of the lens. Fig. 45 shows the chief varieties of lenses—(1) biconvex, (2) biconcave, (3) plano-convex, (4) plano-concave, (5) convexo-concave or meniscus: these names require no further explanation.

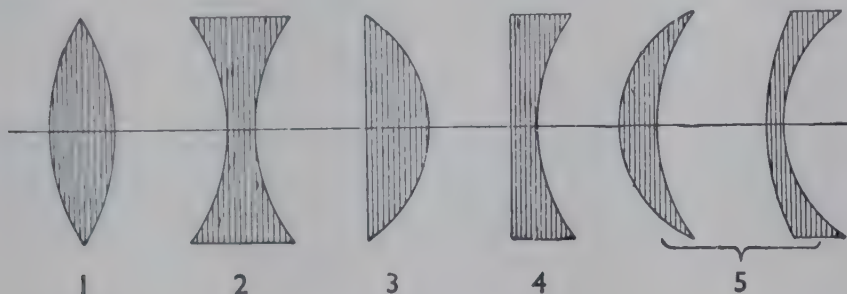


FIG. 45. Types of lenses.

The effect of a biconvex lens upon rays of light meeting it is very similar to what would occur if it were replaced by two prisms set base to base (Fig. 46).

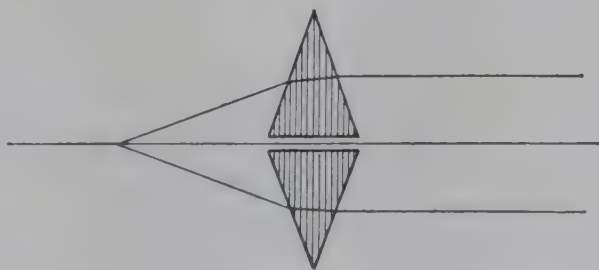


FIG. 46.

If the incident rays are parallel to the axis they will be refracted in such a manner that they all cross the axis in a single point upon the other side of the lens. This point is called the *principal focus* of the lens, and its distance from the lens is called the *focal distance* or *length* of the lens. When the lens has the same medium, such as air, on each side of it, the two principal foci, one on each side of the lens, are situated at equal distances from it. For thin glass lenses of low power the focal distance is equal to the radius of curvature of the two surfaces when these are equally curved. If there is an object a very long distance away from the lens, the rays which come from it are practically parallel. Hence in this case an image of the object

will be formed by the lens at its principal focus ; it will be inverted and very small. If the object is gradually brought nearer and nearer to the lens (Fig. 47) the image will recede farther and farther

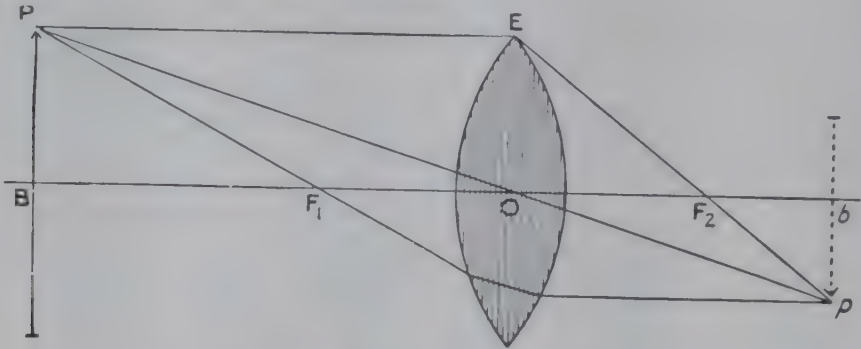


FIG. 47. The ray PE, parallel to the axis, is refracted through the second principal focus F_2 . The ray PF_1 , through the first principal focus, is refracted parallel to the axis. The ray PO, through the optical centre of the lens, is not deflected. The meeting point of any two of these rays gives the situation of p , the image of P .

from it ; from being very small it will grow larger, until, when the object is at the principal focus, the image will have receded to infinity and it will be infinitely large. All the rays coming from an object at the principal focus are therefore parallel to the axis and to each other after refraction. If the object is brought closer to the lens than its focal distance (Fig. 48) it will be found that its image is a

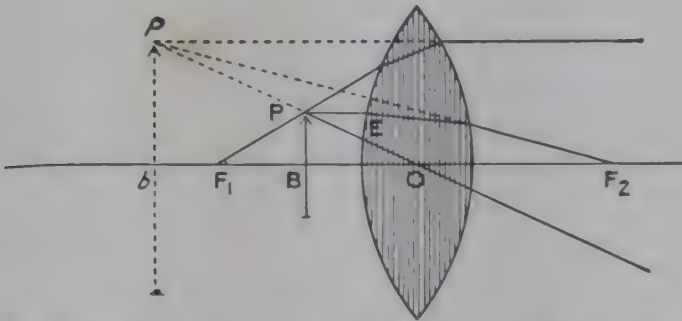


FIG. 48. The description of Fig. 47 applies equally to this figure.

virtual one behind the object, and that it is erect and larger than the object. The positions of the object and image bear a constant relationship to each other and are called *conjugate foci*.

There is a point in the middle of a biconvex lens which is called its *optical centre*. With thin lenses any ray which passes through this point suffers little or no deviation. It is easy to understand why this is so. If PQRS (Fig. 49) is such a ray and tangents are

drawn to the two surfaces at the points Q and R, these two tangents will be parallel to each other. Consequently, the lens acts for such a ray as if it were a plate with parallel sides, and we have already seen that in such a case the emergent ray is parallel to its original direction. If the lens is very thin the refracted ray will be practically continuous with the incident ray.

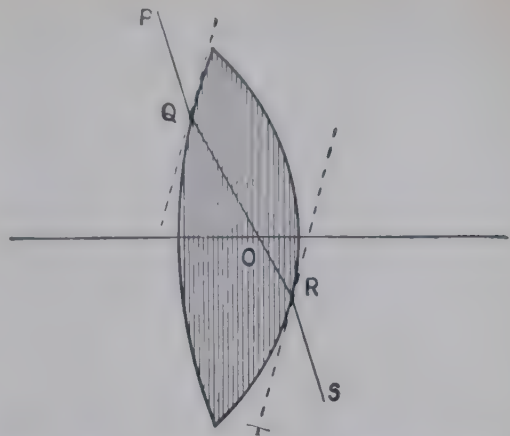


FIG. 49. Properties of the optical centre of a lens.

If we know these facts—that rays passing through the optical centre are not deviated, and that rays passing through the principal focus are parallel to the axis after refraction—we can easily construct the image of an object in any given position.

Thus, in Fig. 47, if PB is an object, the ray PO through the optical centre O will not be deviated; the ray PE parallel to the axis will pass through the second principal focus F_2 ; and the ray PF₁ through the first principal focus will be parallel to the axis after refraction. Hence pb must be the image of PB.

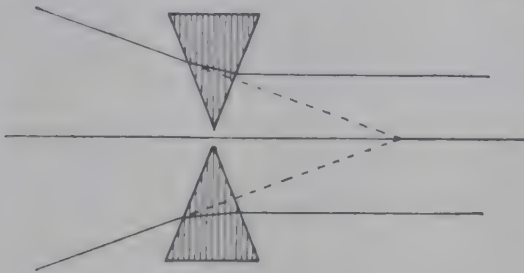


FIG. 50.

The effect of a biconcave lens upon rays of light meeting it is very similar to what would occur if it were replaced by two prisms set apex to apex (Fig. 50).

Here, if the incident rays are parallel to the axis they will be divergent after refraction, and the

amount of divergence of the individual rays will be such that if they are produced backwards they will all cross the axis in a single point upon the

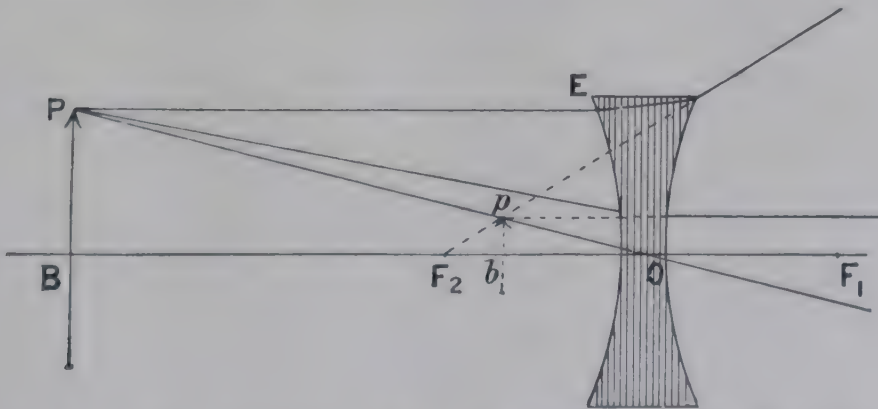


FIG. 51. The description of Fig. 47 applies equally to this figure.

side of the lens from which they came. This and the corresponding point on the other side of the lens are called the principal foci. The biconcave lens also has an optical centre, situated upon the axis within it and having the same properties as in the case of the convex lens. The image of any object formed by a concave lens can be constructed in exactly the same manner as for a convex lens (Fig. 51). It will be found that in every position of the object the image is always virtual, erect and smaller than the object.

Plano-convex and plano-concave lenses act like biconvex and biconcave lenses respectively, but in them the optical centre is on the curved surface at the point where the axis cuts it. Menisci act as convex or concave lenses according to whether the convex or the concave surface has the greater curvature. In them the optical centre is outside the lens.

It will have been noticed that the refractive power of a lens varies inversely as the focal distance, i.e., a lens with a short focal distance will bend the rays more than one with a longer focal distance. It is necessary to have some system of numbering lenses so as to indicate their refractive power. The most convenient system for ophthalmic purposes is that which takes a lens with a focal distance of 1 metre as a standard. Such a lens is said to have a refractive power of 1 *dioptre*.

A lens with a focal length of half a metre will be twice as strong as one with a focal length of 1 metre : the refractive power of such a lens is therefore 2 dioptries. Similarly, a 3 D (3 dioptre) lens has a focal length of one-third of a metre, or 33 cm. ; a 4 D lens, 25 cm. ; and so on. The dioptric power is thus the reciprocal of the focal length ($D = 1/F$). It is important to remember that in this system the standard is a metre, not a centimetre or a millimetre ; otherwise confusion may arise.

Convex lenses are indicated by a plus sign (+), concave by a minus sign (—) before the number.

Cylindrical lenses are also used in ophthalmology ; their nature and use will be considered at a later stage.

We often wish to find out whether a lens is convex or concave, and what is its refractive power. There are several ways of doing this, but the simplest is the following. Hold a convex lens up near the eye and look at distant objects through it ; then move the lens a little from side to side : the distant object will seem to move in the opposite direction to that in which the lens is moved. If we repeat the process with a concave lens the objects seem to move in the same direction as the lens. The reason is to be found in the fact that a convex lens forms an inverted, whilst a concave forms an erect image. If we place two lenses of opposite sign but equal curvature in contact with one another the combination will make a plate with parallel sides : such as plate, as we know, causes no practical deflection of the rays of light. Hence we can determine the strength of a lens by exactly neutralizing it with a lens of the opposite sign.

Let us take a concrete example of a particular lens which we wish to determine. We hold it up and find that distant objects seem to move in the opposite direction to the lens. We know that it is a convex lens. We then put a

weak concave lens in contact with it and repeat the process. We find that with a -2 D lens objects still seem to move in the opposite direction, though not so much. With a -3 D lens there is only a trace of movement, and with a -3.5 D lens there is no movement at all. We conclude that the original lens was $+3.5$ D. In performing this test it is important to have the two lenses as closely in contact as possible, and also to have their centres in contact (Fig. 52).

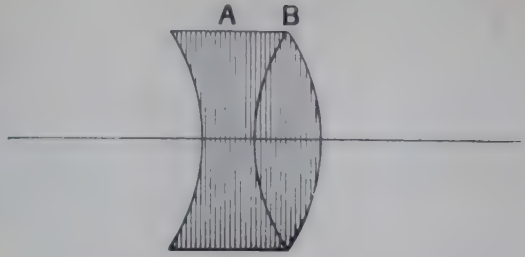


FIG. 52.

If the centre of one lens is higher than that of the other they will obviously not counteract each other exactly. If they are not in contact the result will be either too high or too low.

Systems of Lenses. This leads us to consider what happens when more than one lens is used, the combination forming an optical system. We will confine ourselves to cases where the system is *homocentric*, that is when all the component lenses are centred on a common optic axis in which event the principles already discussed are additively applied.

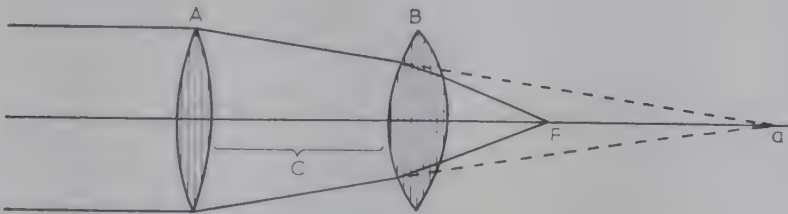


FIG. 53.

When the lenses are in contact the refractive power of the combination (D) is equal to the algebraical sum of the refractive powers of the two lenses (d_1, d_2): i.e., $D = d_1 + d_2$, or $\frac{1}{F} = \frac{1}{f_1} + \frac{1}{f_2}$ where F, f_1, f_2 , are the respective focal distances (Fig. 52).

Suppose, however, that two convex lenses are separated by a distance c (Fig. 53). The lens A will make parallel rays converge towards a , but after a distance c they meet the lens B: hence the convergence of the rays is not expressed by $\frac{1}{f_1}$, but by $\frac{1}{f_1 - c}$. Therefore the combined effect of the lenses.

D , or $\frac{1}{F}$, is now equal to $\frac{1}{f_1 - c} + \frac{1}{f_2}$.

If the second lens (B) is concave (Fig. 54) its effect will be one of divergence, so that it must have a negative sign, and D will now be equal to

$$\frac{1}{f_1 - c} - \frac{1}{f_2}.$$

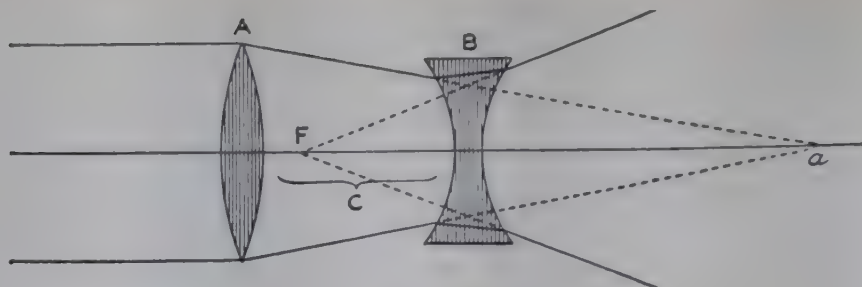


FIG. 54.

It is to be noted that in the formula

$$\frac{1}{F} = \frac{1}{f_1 - c} + \frac{1}{f_2}$$

F is now the posterior focal length, the incident light impinging upon the lens the focal length of which is f_1 , and being directed towards the lens the focal length of which is f_2 . The following formula gives the equivalent focal length (F_e) of the combination, irrespective of the direction of light :

$$F_e = \frac{f_1 f_2}{f_1 + f_2 - c}.$$

CHAPTER 6

ELEMENTARY PHYSIOLOGICAL OPTICS

THE OPTICAL SYSTEM OF THE NORMAL EYE

IN the previous chapter we have shortly outlined the effect of a convex lens in bringing parallel rays of light to a focus (Fig. 47). The optical system of the eye can be deduced from this simple analogy. At first sight the matter would seem to be much more difficult, for instead of a simple convex lens with the same medium (air) on either side, the system comprises a curved optical plate (the cornea), the aqueous humour, the crystalline lens, itself optically complex, and the vitreous body. The object of this more complicated arrangement is to shorten the focal distance of the system, so

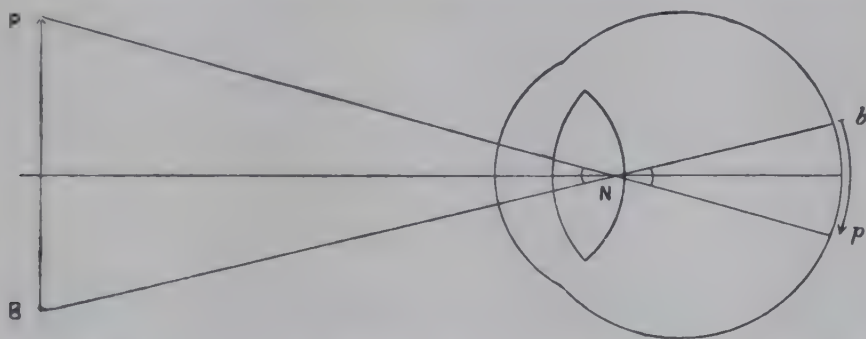


FIG. 55. The formation of retinal images in the schematic eye. The image pb of an object PB is formed by drawing lines from P and B through the nodal point (N). PNB or pNb represents the visual angle.

that the eye may be smaller and more compact. Moreover, the medium in front is air, while behind the lens there is the vitreous which has a higher refractive index. Fortunately great simplifications can be made. The cornea has almost the same refractive index as the aqueous, which is also equal to that of the vitreous. The anterior surface of the cornea may be regarded as nearly spherical, the radius of curvature being 8 mm. The centres of curvature of the cornea and the two surfaces of the lens are all on the same straight line, which is called the *optic axis*. Indeed, from the optical point of view, with little loss of accuracy, the entire system can be regarded as one lens with one optical centre (the *nodal point*, N) which lies in the posterior part of the crystalline lens (the *schematic eye*) (Fig. 55).

Since the rays enter and leave the refracting system through media of different optical density, the anterior and posterior focal distances are different, the former being about 15 mm. in front of

the cornea, the latter about 24 mm. behind it. It follows that if parallel rays fall upon the cornea they will be brought to a focus 24 mm. behind it. Since the small bundles of rays which enter the pupil may be considered parallel, the image formed by the eye of distant objects will be at this point, which in the normal eye lies on the retina. Hence the normal eye in its condition of rest is so constituted that distant objects form their images upon the retina (Fig. 56).

The optic axis, produced backwards to meet the retina, cuts it almost exactly at the fovea centralis. Hence, any distant object on the prolongation forwards of the optic axis will have its image at the fovea, which is the best spot for distinct vision. Here, just as with a convex lens, the image is inverted; it is re-inverted psychologically in the brain.

It is easy to find the size of the retinal image which any external object will form, since the nodal point (N) corresponds to the optical centre of a convex lens. As in the case of lenses, any ray which passes through this point will not be appreciably deflected. If, therefore, there is an object PB (Fig. 55) in front of the eye, the size of its retinal image pb is found by joining the extremities of the object and the nodal point and producing these lines until they meet the retina. The lines will enclose an angle, PNB, which is called the *visual angle*, the angle subtended by the object at the nodal point. It is of course equal to the angle pNb , which is subtended by the retinal image at the nodal point.

THE OPTICAL SYSTEM OF THE ABNORMAL EYE

In some eyes the retina is not situated in exactly the right place for the images of distant objects to be clearly focused upon it. It

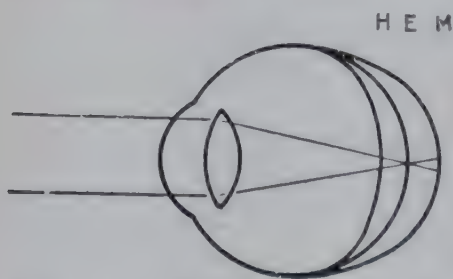


FIG. 56. Emmetropia, hypermetropia and myopia. In emmetropia (E), parallel rays of light are focused upon the retina. In hypermetropia (H), the eye is relatively too short; in myopia (M), it is too long.

may be too far forward, or too far back (Fig. 56); in the former case the eye is said to be *hypermetropic*, in the latter *myopic*. If we consider the effect upon parallel rays we shall see that in the hypermetropic eye they have not yet come to a focus, whereas in the myopic eye they have not only come to a focus but have commenced to diverge. In each case a blurred image will be formed upon the retina, and vision will be impaired. Such conditions are called *errors of refraction* or *ametropia* (α , privative, $\mu\acute{\epsilon}\tau\rho\omicron\nu$, measure; not according to measure).

In contradistinction to hypermetropia and myopia the normal condition is called *emmetropia*. The condition of an eye, whether emmetropic, hypermetropic or myopic, is called its *refraction*, or more accurately its *static*

refraction, since the term applies to the eye at rest. When, as commonly happens, the refractions of the two eyes are different, the condition is called *anisometropia* (α , privative ; ἴσος , equal ; μέτρον , measure).

It has already been stated that in optics the direction of the rays is reversible. If we imagine a minute point on the emmetropic retina to be luminous, it will give out rays which will diverge in all directions. The rays which pass through the pupil out of the eye will have to submit to exactly the same optical deviations as the parallel rays falling upon the cornea when they passed into the eye and will therefore be parallel to each other when they leave the eye.

Suppose, however, that the eye is hypermetropic because it is too short (Fig. 56). The rays coming from a point on the retina will be relatively more divergent than the corresponding rays of the emmetropic eye before they fall upon the back of the lens. (Compare the effect of placing an object closer to a convex lens than its principal focus, Fig. 48.) They will therefore still be divergent when they leave the eye, though of course not so divergent as when they were passing through the vitreous. In fact, their direction will be the same as if they

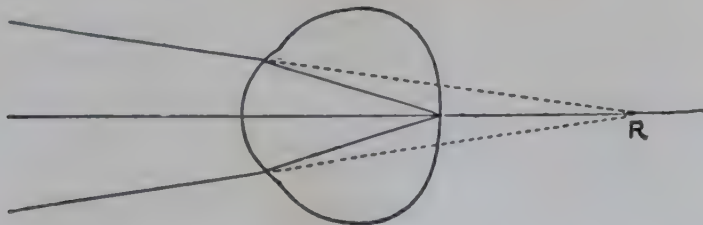


FIG. 57. Hypermetropic eye. Rays from a point on the retina are divergent when they emerge from the eye, as if they came from the point, R, behind the eye.

came from a point behind the eye. The nearer the retina is to the lens, the more divergent they will be, and the nearer to the back of the eye will be the point from which they seem to come. This virtual point (R) behind the eye is called the *remote* or *far point* of the eye. The point on the retina and this point behind the eye are really *conjugate foci* (Fig. 57).

Suppose now that the eye is myopic because it is too long (Fig. 56). The rays coming from a point on the retina will be relatively less divergent than the corresponding rays in the emmetropic eye before they fall on the back of the lens. (Compare the effect of placing an object farther away from a convex lens than its principal focus, Fig. 47.) The refractive media in front will therefore cause them to converge more than in the emmetropic eye. They will thus be convergent when they leave the eye, and will cross at a point (R) somewhere in front of the eye (Fig. 58). The farther the retina is from the lens, i.e., the higher the degree of myopia, the more convergent they will be, and the nearer to the front of the eye will be the point where they cross. This point is again the conjugate focus to the point of the retina, but in this case it is a real point. It is also called the *remote* or *far point* of the eye.

We have seen that in the emmetropic eye the emergent rays are parallel to each other. Since parallel rays meet at infinity, the far point of the emmetropic eye is at infinity.

We have seen also that in every case the far point and a point on the retina are conjugate foci. Using the principle of the reversibility of rays, any object situated at the far point of any eye will have a sharp image upon the retina (Fig. 58). Thus the emmetrope sees only distant objects clearly with his eyes at rest, since the rays

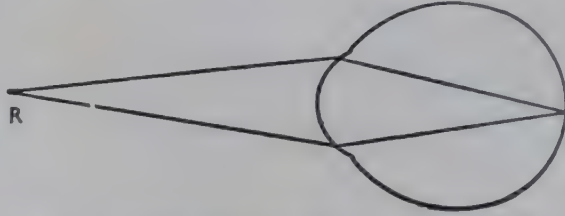


FIG. 58. Myopic eye. Rays from a point on the retina are convergent when they emerge from the eye, so that they cross at a real point, R, in front of the eye.

from such objects are nearly parallel ; for practical purposes this applies to objects more than six metres away. On the other hand, a patient with myopia can only see things which are near ; he is "short-sighted." He can see things at a distance better if he screws up his eyes because he thus makes a narrow slit through which to look, and this slit acts in the same way as the hole in the cardboard in the experiment illustrated in Fig. 37. The term myopia originated in this peculiarity ($\mu\acute{\nu}\epsilon\iota\nu$, to shut ; $\omega\psi$, the eye). Again, a patient with hypermetropia can see neither distant nor near objects clearly with his eyes at rest, since the far point is virtual, and it is impossible to place an object at its situation. We shall see later that when young he is better off than the myope since he can alter his refractive power by accommodation.

Errors of refraction may be due to causes other than axial shortening or lengthening of the eye (*axial ametropia*). They may be due to alterations in the refractive indices of the media, or to alterations in the curvatures of the refractive surfaces ; ametropia due to these causes is called *index* or *curvature ametropia* respectively. Index ametropia is rare, apart from changes in the refractivity of the lens.

Curvature ametropia has a special importance because it is the cause of another very troublesome error of refraction, called *astigmatism*. In most eyes the areas of the refractive surfaces uncovered by the pupil and used in vision are very nearly spherical. Sometimes, however, they are not. In most of these cases it is the cornea which is at fault, and the error is generally of such a nature that its surface is flatter from side to side than it is from above downwards, perhaps because the pressure of the lids on the globe tends to squeeze it above and below.

When the cornea has its direction of greatest and least curvature at right angles to one another, the condition is called *regular astigmatism*. In the commonest form when the vertical meridian is the more curved, the condition is generally called regular astigmatism "according to the rule"; the reverse is said to be "against the rule," but not infrequently the axes are oblique. Often, as after ulceration, the surface of the cornea is irregular so that the rays of light are refracted irregularly without any symmetry and different groups form foci in various positions. This is called *irregular astigmatism*: it cannot be corrected, and can only occasionally be improved by lenses.

Although astigmatism is chiefly due to faulty curvature of the cornea, in some cases there is also lenticular astigmatism. This is not generally due to unequal curvature of the surfaces, but to slight tilting of the lens, so that the incident rays fall upon it obliquely. If we look through a tilted glass lens at printed matter we shall see that the letters become distorted and elongated in one direction; this is a form of astigmatism. The astigmatism of the crystalline lens is generally of such a nature that it tends to counteract the corneal astigmatism although sometimes it adds to the effect.

A regularly astigmatic surface is said to have a *toric* curvature. In it the more curved meridian will have more refractive or convergent power than the less curved: hence if parallel rays fall upon such a surface the vertical rays will come to a focus sooner than the horizontal. The rays after refraction will be perfectly symmetrical when referred to the vertical and horizontal planes but they will have two foci. The whole bundle of rays is called *Sturm's conoid*, and the distance between the two foci is called the *focal interval* of Sturm. It is difficult to represent this conoid on a plane surface (Fig. 59), but we can see what sections of the bundle or pencil of rays would look like at different distances from the refractive surface (Fig. 59, A—G).

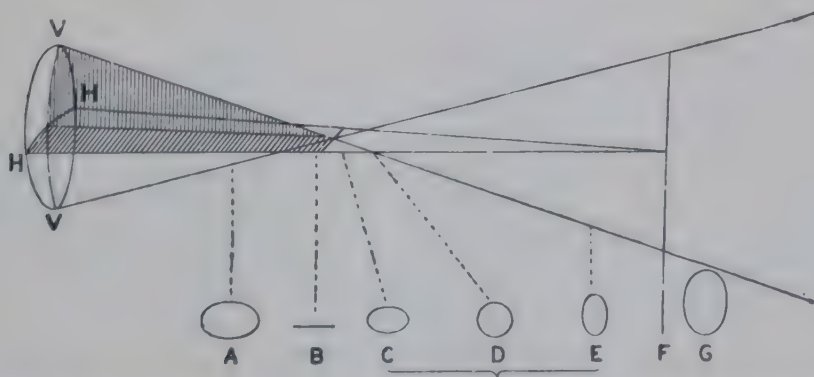


FIG. 59. Sturm's conoid. V V, vertical meridian of refracting surface, more curved than H H, the horizontal meridian. A, B, C, D, E, F, G, sections of conoid. From B to F is the focal interval of Sturm. D shows the circle of least diffusion.

At A the section will be a horizontal oval or oblate ellipse, because the vertical rays are converging more rapidly than the horizontal. At B the vertical rays have come to a focus, while the horizontal are still converging : the section will be a horizontal straight line. At C, D, and E the vertical rays are diverging and the horizontal are still converging. At one place in this focal interval there will be a spot (D) where the vertical rays have diverged from the axis exactly as much as the horizontal rays have converged towards it. Here the section is a circle, which is called the *circle of least diffusion*. At F the horizontal rays come to focus while the vertical are diverging : the section will be a vertical straight line. Beyond this point, as at G, both sets of rays are diverging, and the section will always be a vertical oval or prolate ellipse.

If the retina is situated at any of these points of section, it is obvious that the retinal image will always be blurred ; it is because the rays never come to a focus in a single point that the condition is called *astigmatism* (ἄ, privative, στίγμα, a point). If the retina cuts the conoid at A, where none of the rays has come to a focus, every meridian will be in the same condition, though in different degree, as in the axial hypermetropic eye : this condition is therefore called *compound hypermetropic astigmatism*. If the retina is at B the vertical meridian will be in the condition of an emmetropic eye, while the horizontal will still be in the condition of hypermetropia : this condition is called *simple hypermetropic astigmatism*. At C, D, and E the vertical meridian will be in the condition of a myopic, and the horizontal still in that of a hypermetropic eye : this is called *mixed astigmatism*. At F the vertical meridian is still myopic, whilst the horizontal is in the same condition as in an emmetropic eye : this is *simple myopic astigmatism*. Beyond F, as at G, both meridians are in the condition of an axial myope, the rays having crossed in the vitreous : this is *compound myopic astigmatism*. All these positions of the retina are met with in actual practice, although there is often a combination of axial and curvature defects.

Distant vision is often found to be surprisingly good with relatively high degrees of mixed astigmatism, probably because the circle of least diffusion falls on or near the neuro-epithelium of the retina.

THE CORRECTION OF AMETROPIA WITH LENSES

It is obvious that, in hypermetropia, if we give the rays the requisite amount of convergence before they enter the eye by placing a convex lens in front of it, they will be brought to a focus upon the retina (Fig. 60). This is what is done by means of spectacles. The refractive or convergent power of a convex lens is the reciprocal of its focal distance. Hence in hypermetropia of 1 D, a convex lens of 1 D or 1 metre focal distance placed in contact with the cornea, acting in combination with the refractive power of the eye, would bring the rays to a focus on the retina. But lenses are only rarely worn in contact with the cornea. If the lens is placed 20 mm. in front of the cornea its focal length will have to be 1,020 mm. instead of 1,000 mm., but this small difference is negligible, and we are

accustomed to measure errors of refraction by the strength of the lens which is required when it is placed in the ordinary position of a spectacle lens.

Similarly in myopia, if we give the rays the requisite amount of divergence before they enter the eye they will be brought to a focus upon the retina. We do this by placing a concave lens in front of

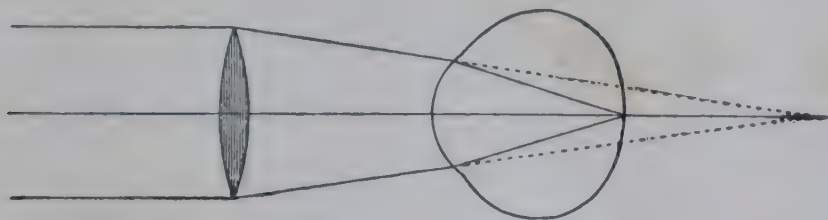


FIG. 60. Hypermetropic eye. Parallel incident rays brought to a focus on the retina by means of a suitable convex lens.

the eye (Fig. 61). Here we should want a -1 D lens in contact with the cornea to correct a myopia of 1 D, i.e., an eye the far point of which is 1 metre in front of the eye. If the glass is worn about 20 mm. in front of the eye it will have to be somewhat stronger, i.e., it will have to be a focal distance of 980 mm. instead of 1,000 mm.

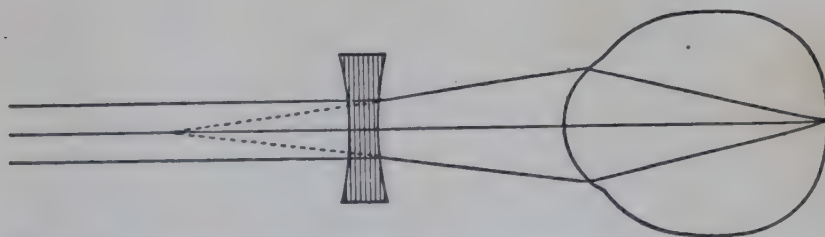


FIG. 61. Myopic eye. Parallel incident rays brought to a focus on the retina by means of a suitable concave lens.

There is an advantage in having the correcting lenses in axial ametropia in the position of the anterior focus of the eye, because in these conditions the size of the retinal image is the same as if the eye were emmetropic (Figs. 62, 64). The anterior focus is about 15 mm. in front of the eye. The optician aims at placing the optical centre of the spectacle lens 12–13 mm. from the cornea. We have already discovered that the farther the lens is from the eye, the convex lens in hypermetropia has to be weaker, and the concave lens in myopia stronger. There is also an effect on the size of the retinal image (see Figs. 63, 65). If the lens is more than 15 mm. from the cornea the retinal image in hypermetropia is larger, and in myopia smaller than the emmetropic image. The increase in size in hypermetropia is advantageous, but the diminution in myopia is a disadvantage, especially in very high degrees. Consequently, in the latter the spectacles ought to

be made to fit as closely to the eyes as possible, the eyelashes being cut short if necessary.

In astigmatism we must obtain some means of affecting one set of rays more than the other. This means is found in cylindrical lenses.



FIG. 62.

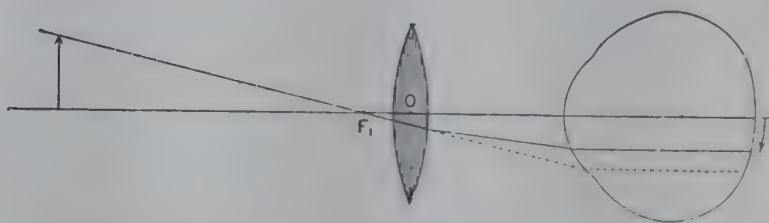


FIG. 63.

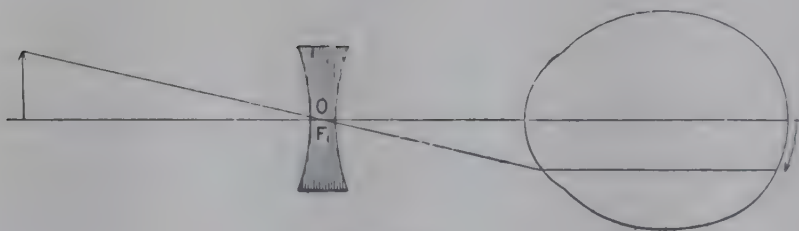


FIG. 64.

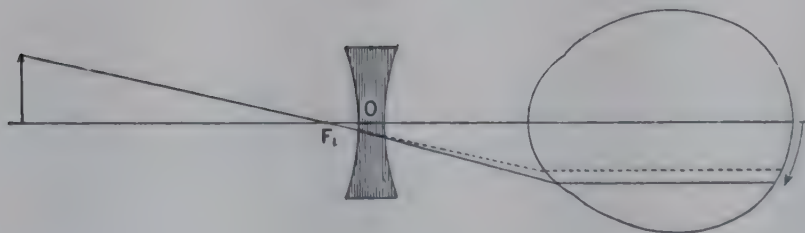


FIG. 65.

FIGS. 62-65. Effect of correcting lenses upon the size of the retinal image. In Figs. 62, 64, where the optical centre of the lens, O , coincides with the anterior focal point of the eye, F_1 , the size of the retinal image is the same as in emmetropia. When the lens is closer to the eye than the anterior focal distance of the eye the size of the retinal image is diminished (convex lens, Fig. 63) or increased (concave lens, Fig. 65).

Suppose CDEF is a cylinder of glass (Fig. 66) : AB is called the axis of the cylinder. If a slice is cut off the cylinder by a plane parallel to the axis, it would form a cylindrical lens. Fig. 67 gives representations of a convex and a concave cylinder. The direction *ab* is called the axis of

the cylinder, since it is parallel to the axis of the original cylinder from which the slice may be supposed to have been taken. It is important not to confuse the axis of a spherical and the axis of a cylindrical lens, as they are totally different things. The axis of a cylinder has just been described : the axis of a spherical lens is the line joining the centres of curvature of the two surfaces.

Parallel rays falling upon a cylindrical lens will be affected in different ways. In the direction of its axis it is simply a plane lamina with parallel sides, so that it will have no effect upon the rays. In the direction at right angles to its axis it is spherical on one side and plane on the other : it will therefore

act exactly like a plano-convex or a plano-concave lens, i.e., it will make the rays either converge or diverge (Fig. 68). If a convex cylinder is held between a point of light and a screen, a position can be found for the screen

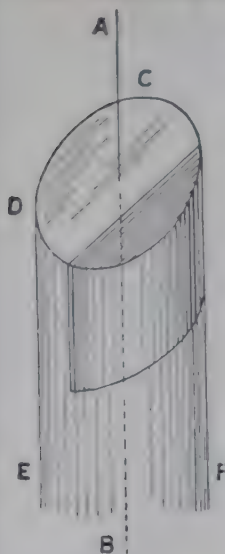


FIG. 66.

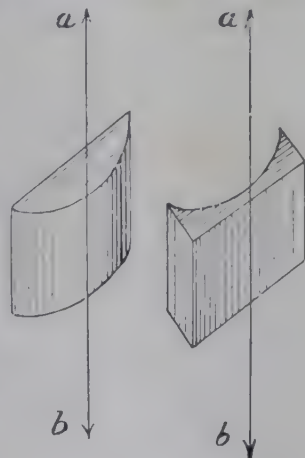


FIG. 67.

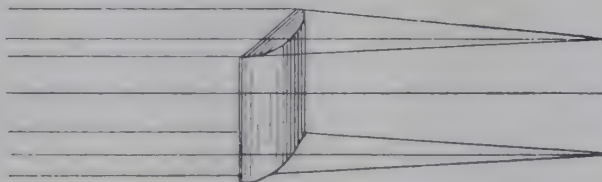


FIG. 68. Refraction of parallel rays through a plano-convex cylinder.

such that a sharp bright line is thrown upon it (Fig. 69) : this is the *focal line* of the cylinder.

It is to be noted that the line is in the direction of the axis of the cylinder. If another convex cylinder of the same strength were held with its axis at right angles to the first, it would obviously form a focal line perpendicular to the first

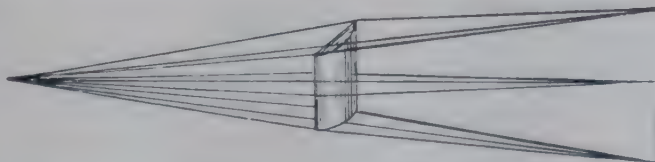


FIG. 69. Refraction of divergent rays from a point of light through a plano-convex cylinder.

focal line. If the two cylinders are put in contact with their axes at right angles, all the rays after refraction must pass through both lines. The only place where they can go through both lines is where the lines intersect. Hence we see that two cylindrical lenses of equal strength, placed in contact with their

axes at right angles, act exactly like a convex spherical lens of the same strength as either of the cylinders.

Optical Aberrations. As in all optical systems in practical use, the eye is by no means optically perfect; the lapses from perfection are called *aberrations*. To a large extent, however, they affect the peripheral rays and are thus eliminated by the iris which acts like the diaphragm of any ordinary optical system, such as a photographic camera or a microscope. In discussing the effects of spherical mirrors in reflecting, and of spherical surfaces in refracting rays of light, we said that in each case they were all brought to a focus in a single point. This is really only an approximation which is sufficiently accurate for rays close to the axis. In a convex spherical lens, for instance, only parallel rays near the axis meet at

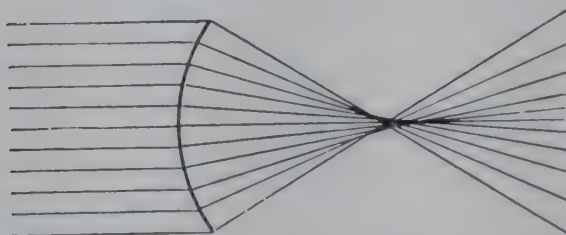


FIG. 70. Spherical aberration.

the principal focus; rays farther away from the axis, however, are refracted too much, so that they cut the axis nearer the lens than the principal focus thus causing a blurring of the edges of the image (*spherical aberration*) (Fig. 70). A diaphragm cutting off these peripheral rays would prevent the blurring. In the eye the surfaces are not spherical, especially near the periphery, so that much more aberration is liable to occur, but the iris reduces the effects to a minimum.

There is also another form of aberration due to the imperfect refraction at spherical surfaces. White light is made up of all the colours of the spectrum. The component rays are refracted differently, the short violet rays most, the long red least. Hence there is a tendency for the white light to be split up into its components, in which case the image will have a coloured edge (*chromatic aberration*); this effect in the eye, however, is small.

Several other aberrations occur which are relatively unimportant; their effect, however, may be increased and others introduced, particularly affecting oblique and peripheral rays, when the optical system is complicated by spectacles.

ACCOMMODATION

We have to consider now how a person with normal sight can see not only distant objects, but also near ones. If an object is situated

near the eye, as at ordinary reading distance (about 30 cm.), the divergence of the rays which it emits cannot be neglected. Since the converging power of the refractive media of the emmetropic eye is only strong enough to make parallel rays come to a focus on the retina, it is obvious that divergent rays falling upon the cornea will not have come to a focus (Fig. 71). The necessary

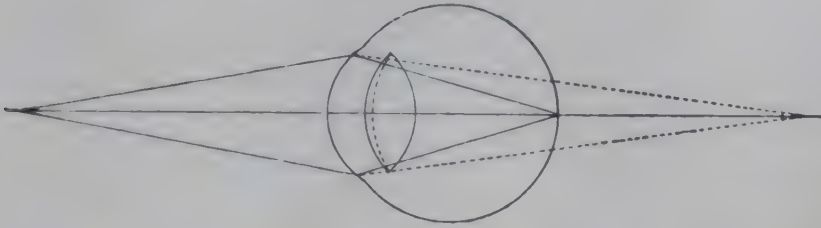


FIG. 71. Effect of accommodation. The dotted lines show the curvature of the anterior surface of the lens and the course of rays with the eye at rest (static refraction). The solid lines show the curvature of the anterior surface of the lens and the course of rays with active accommodation (dynamic refraction).

increase in their convergence is accomplished by augmenting the refractive power of the crystalline lens by increasing the curvature of its surfaces in the act of *accommodation*.

The curvature of the surfaces of the lens at rest in the eye is approximately spherical, the radius of curvature of the anterior surface being 10 mm., that of the posterior surface 6 mm. In accommodation, the curvature of the posterior surface remains almost the same, but the anterior surface changes so that in strong accommodation its radius of curvature becomes 6 mm. The eye in this condition, which is called its *dynamic refraction*, has a much increased converging effect upon the incident rays.

The mechanism by which this change in the curvature of the lens is brought about has excited much controversy. It would seem that the lens itself has a considerable amount of elasticity which determines its normal non-accommodated form (Fig. 72). The capsule, however, is more elastic, and when the ciliary muscle contracts the ciliary body approaches the lens, thus slackening the zonule so that the capsule, relieved of tension, is able to mould the lens into its accommodated form. The peculiar shape assumed by the lens thus deformed may be due to the peculiar configuration of the capsule which is thicker behind the iris than in the central area. The shape of the lens at any one time is thus the result of a balance between its own elasticity and that of its capsule.

Our control over the ciliary muscle, though involuntary, is very delicate, so that all distances up to quite close to the eye can be accurately focused. The nearest point at which small objects can be clearly distinguished is called the *near point*, or *punctum proximum*. At this point accommodation is exerted to its maximum,

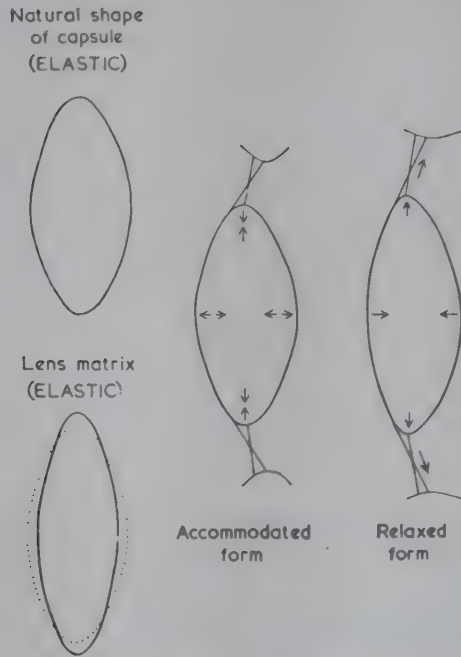


FIG. 72. The changes in the lens with accommodation. During accommodation the elastic capsule imposes its natural conoidal shape on the elastic lens substance which resists the former (after Weale).

the lens capsule is as slack as it is possible to make it, and an object closer to the eye could only be seen clearly by using a convex lens.

It has been shown that the *far point* or *punctum remotum* of the eye varies according to its static refraction, that is, whether it is emmetropic, hypermetropic, or myopic. The near point also varies with the static refraction, and again with the age of the patient, the reason being that the lens becomes less plastic as age advances. We have stated that the lens is a mass of epithelium of which the central part is the oldest; as it gets older the central cells become tougher and more compressed, thus forming a relatively hard nucleus. The nucleus is less plastic than the younger cortex and, as age advances, more and more of the fibres become converted into nucleus. Consequently the lens tends to respond less and less to changes in tension of the capsule. Thus, a child of ten is able to see a small object clearly when it is only 7 cm. from the eye, while a person of thirty years of age may not see clearly at less than 14 cm.

Normally the ciliary muscle has a considerable amount of tone which cannot be relaxed so that the full degree of hypermetropia is only apparent when this muscle is paralysed by a cycloplegic drug. This portion of the total hypermetropia which can only be revealed under atropine is called the *latent hypermetropia*. The remainder, which is normally uncorrected, is called the *manifest hypermetropia*. The sum of the two gives the *total hypermetropia*. Of the manifest hypermetropia, that part which can be relaxed by accommodation

is termed *facultative*; that which cannot be thus relaxed, *absolute*. In extreme youth nearly all the hypermetropia is latent: the lens is so resilient that it is impossible to prevent it responding to the slightest stimulus. As the lens becomes less plastic more and more of the hypermetropia becomes manifest, until finally, when accommodation disappears entirely, all the hypermetropia is manifest. The older the patient, therefore, the more nearly the manifest hypermetropia represents the total amount.

We have pointed out that the refractive power of a lens in dioptries is the reciprocal of its focal distance measured in metres and that the same method is applied to measure the static refractive powers of the eye. Applying the same method to the dynamic refractive power, the child of ten, whose near point is 7 cm. from his eye, has a refractive power of $100/7 = 14$ D, and a man of thirty, whose near point is 14 cm. from his eye, has a refractive power of $100/14 = 7$ D.

By this means we can obtain a general rule for indicating the amount or *amplitude of accommodation*, not only of emmetropic but also of hypermetropic or myopic eyes. This is given by the formula $A = P - R$, which states that the amplitude of accommodation (A) is equal to the refractive power of the eye when fully accommodated (P) (i.e., the reciprocal of the distance of the near point in metres) less the refractive power of the eye at rest (R) (i.e., the reciprocal of the distance of the far point in metres).

Thus, the emmetropic child of ten has an amplitude of accommodation of $100/7 - 1/\infty = 14 - 0 = 14$ D. Similarly in the case of an emmetrope whose near point is 12.5 cm. from his eye, the amplitude of accommodation (A) = $1,000/125 - 1/\infty = 8$ D. Again, a myope of 2 D whose near point is 8 cm. in front of his eye will have an amplitude of accommodation (A) = $100/8 - 2 = 10.5$ D. Again, in the case of a hypermetrope of 3 D whose near point is 12.5 cm. from his eye, the far point is behind the eye and distances measured in this direction must have the opposite sign to those measured in front of the eye. Hence $A = 1,000/125 - (-3) = 8 + 3 = 11$ D.

The numbers given by these calculations for the amplitude of accommodation give the strength of the convex lens which would have to be placed in contact with the cornea in order that the near point might be brought to the required distance without using the accommodation. Several interesting facts come to light from the calculations. Thus a hypermetrope of 3 D has to exert 11 D of accommodation in order that he may see clearly at 12.5 cm., while an emmetrope has to exert only 8 D of accommodation to bring about the same result. We see, then, that the hypermetrope has to exert an amount of accommodation equivalent to the amount of his hypermetropia in order to focus parallel rays upon his retina and see distant objects clearly. Again, the myope of 2 D, whose far point is half a metre, or 50 cm., from his eye, can see clearly at that distance without accommodating, but he has to exert 10.5 D of accommodation in order that he may see clearly at 8 cm. from his eye. This patient, then, has to exert nearly as much accommodation to alter his points of clear vision from 50 cm. to 8 cm., i.e., through 42 cm., as a hypermetrope of 3 D has to employ in order to move his point of distinct vision from infinity up to 12.5 cm. We

see, therefore, that the *range of accommodation*, that is, the distance between the far point and the near point, is not always the same for a given amplitude.

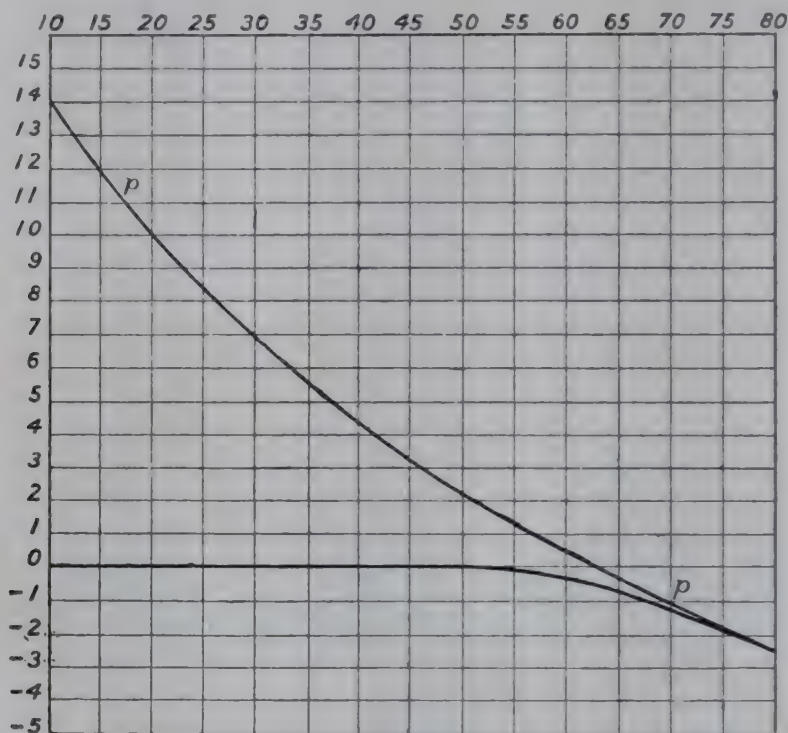


FIG. 73. Chart of static (lower curve) and maximum dynamic (upper curve) refraction at various ages (Donders). Abscissa, ages; ordinate, accommodation in dioptres.

The effect of age upon the static and dynamic refraction is given in Fig. 73, which is compiled from a large number of statistics. From this graph we see that even the far point alters in advanced age. After about fifty years the eye tends to become hypermetropic, so that at eighty it has about 2.5 D of hypermetropia; this is due to an alteration in the refractive index of the lens so that it has a weaker converging power.

The refractive indices of the successive layers of the lens increase from the periphery towards the nucleus. The effect is twofold: it tends to correct aberration by increasing the convergence of the central rays, and the total refractive index of the whole lens is increased, becoming greater than the refractive index of the nucleus. The lens may be looked upon as a central biconvex lens encapsulated in two menisci (Fig. 74); these act as concave lenses because the curvature of the nucleus is greater than that of the periphery of the lens. Hence they tend to counteract the effect of the central lens, but not so much as if their refractive indices were the same. In old age the index of the peripheral layers usually increases, so that the total refractive index of the lens becomes less, and the eye becomes hypermetropic.



FIG. 74.

If we turn our attention to the curve of the near point in Fig. 73 we see that the amplitude of accommodation gradually diminishes throughout life. Since we are accustomed to hold books for reading at about 25 cm. from the eye, in order to see clearly we must exert $100/25$ or 4 D of accommodation which is all that an emmetrope has available at a little over forty years of age ; he will still be able to see clearly at 25 cm., but not closer. If he is about forty-six he will have only 3 D of accommodation left and will have to hold his book farther off, at $100/3$ or 33 cm., a disability which will increase as age advances. This is the condition which is called *presbyopia* (*πρέσβυς*, old).

It is a common error to think that presbyopia is a condition which commences at about forty-five years of age in emmetropes, and earlier in hypermetropes. It is important to remember that the condition has been increasing throughout life and first becomes troublesome when the near point of the eye has receded so far that it is beyond comfortable reading or working distance.

There are two other phenomena which occur with accommodation, one affecting the iris, the other the direction of the eyes. In order that we may see a near object with both eyes they must each turn inwards or *converge*. The amount of convergence, like the amount of accommodation, depends upon the distance of the object so that there is a close relationship between accommodation and convergence. We shall have more to say upon this subject when we consider the various forms of squint.

When we accommodate for a near object the pupil becomes smaller, or contracts. Experiment has shown that this movement of the iris is associated with the accompanying act of convergence rather than with accommodation. This contraction of the pupil during accommodation helps to diminish aberration by cutting out the peripheral parts of the lens, increases the depth of focus and compensates for the relative increase of light entering the eye from near objects. It may be noted that in accommodation the ciliary muscle contracts equally all round the circumference and equally and simultaneously in the two eyes, so that the activity can correct neither astigmatism nor anisometropia.

CHAPTER 7

THE DETERMINATION OF THE REFRACTION

In determining the refraction of the eye the best routine is, first, to estimate the condition objectively, and then to verify and adjust these findings by subjective tests. The objective methods commonly employed are retinoscopy and the use of the refractometer or (for the corneal astigmatism) the keratometer.

RETINOSCOPY

The Theory of Retinoscopy. *Retinoscopy* or, more correctly, *skiascopy* or *the shadow test*, is the most practicable method of estimating the condition of the refraction objectively. It depends upon the fact that when light is reflected from a mirror into the eye the direction in which the light travels across the pupil varies with the refraction of the eye. The light seen in the pupil is the blurred image of the illuminated area of the fundus as seen by the observer when he accommodates for the observed pupil ; it is bordered by a shadow representing the image of the edge of the illuminated area. If the light is thrown into a myopic eye from a concave mirror at a distance of 1 metre and the mirror is tilted in any direction the light, or, what is easier to observe, the shadow, moves across the pupil in the same direction (Fig. 75). If a plane mirror is used, the other conditions remaining the same, the shadow will be seen to move in the opposite direction to the movement of the mirror. If the

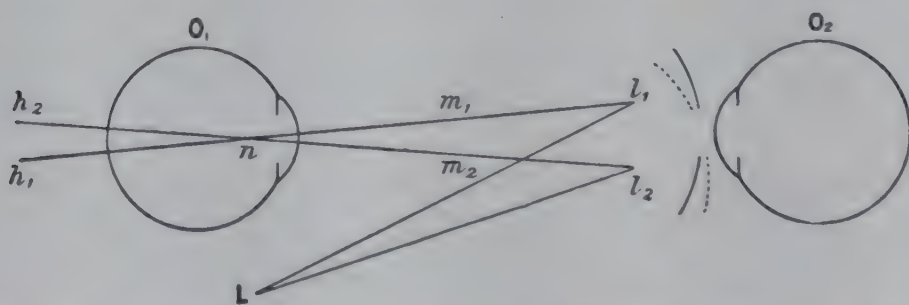


FIG. 75. Diagram of retinoscopy with a concave mirror. O_1 , the observed eye ; O_2 , the observer's eye. The image of the source of light (L) is formed at l_1 (the immediate source of light) by the mirror. If O_1 is hypermetropic a virtual image of l_1 is formed on the line $l_1 n$, passing through the nodal point n , as at h_1 . If O_1 is myopic a real inverted image is formed as at m_1 . If the mirror is tilted downwards, as shown by the dotted line, l_1 moves to l_2 , h_1 to h_2 , and m_1 to m_2 . This shows that the shadow moves in the opposite direction in hypermetropia and the same direction in myopia.

eye is hypermetropic the direction in which the shadow moves is the opposite to that with the myopic eye. If the eye has one dioptré of myopia no shadow will be visible ; the pupil will be either completely illuminated or completely dark. The method therefore consists in placing lenses in front of the eye until no shadow is seen ; if the surgeon is 1 metre away from the patient the combination of the optical system of the patient's eye and the lenses is equal to 1 dioptré of myopia.



FIG. 76. Showing the course of incident rays and field of illumination of the fundus in hypermetropia. l_1 forms a virtual image at λ_1 , l_2 at λ_2 . The field of illumination is determined by the pupil of O_1 .

A simple optical explanation is as follows. Rays from a point of light in front of the eye illuminate a circular area of the fundus, varying in size according to the refraction of the eye (Fig. 76). If the point of light moves upwards, the light on the retina will move downwards.

In the hypermetropic eye the rays reflected from the illuminated area will be divergent, as if they came from a point behind the eye. This far point, corresponding to the illuminated area, will move in the same direction, i.e., downwards. If an observer, placed in front of the eye, looks towards a point of light situated at the position of the far point, but accommodates for the position of the observed pupil, he will see a circle of light with a blurred margin, not a point, because he is not accommodating accurately for the far point. When the illumination on the retina moves down, the circle of light which the observer sees will appear to move down also (Fig. 76).

In a highly myopic eye, on the other hand, the rays of light reflected from the illuminated area on the fundus will be convergent and will cross at a real point in front of the eye. This far point, corresponding to the illuminated area, will move upwards when the illuminated area moves downwards. If an observer placed in front of the eye and farther from it than the far point, looks towards the far point but

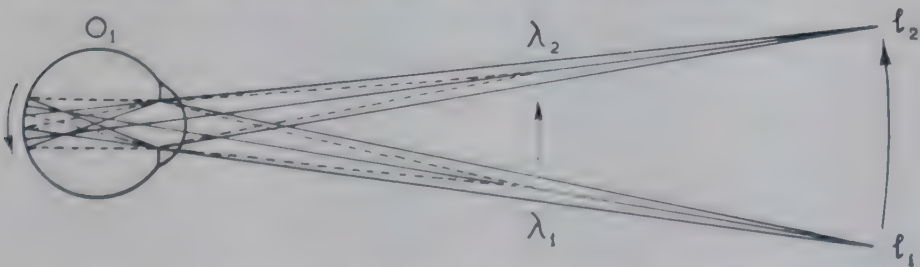


FIG. 77. Showing the course of incident rays in myopia.

accommodates for the observed pupil, he will see a circle of light with a blurred margin. When the illumination on the retina moves down, the circle of light which the observer sees will move up, i.e., in the opposite direction to the movement in the case of the hypermetropic eye (Fig. 77).

If the observer's eye is one metre in front of the observed eye, and the latter has 1 D of myopia, the far point of the observed eye will be at the situation of the observer's eye (Fig. 78). In this case a very slight movement of the light on the observed fundus will throw the image at the far point off the observer's eye altogether ; in other words,

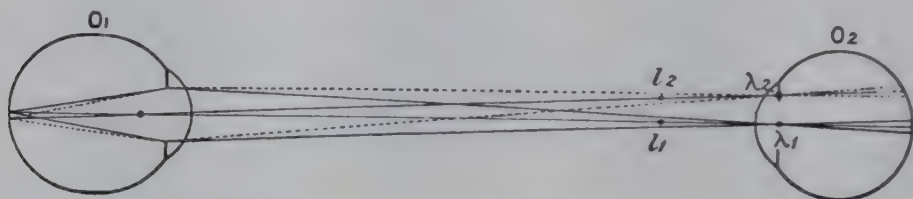


FIG. 78. Showing the course of the emergent rays at the point of reversal. So long as λ_1 is in the pupillary area of O_2 , the pupil of O_1 appears uniformly illuminated, and there is no shadow. Directly λ_1 passes to λ_2 the whole of the light is cut off, so that the pupil of O_1 becomes completely dark.

the observed pupil will appear to be completely bright or completely dark.

If, again, the observed eye is emmetropic, its far point will be at infinity ; we may regard it as being infinitely far behind the observed eye. Here, again, there will be scarcely any shadow, although in reality there is a very faint shadow moving in the same direction as for the hypermetropic eye.

The type of mirror used is an entirely subsidiary matter ; it merely determines the direction of movement of the immediate source of light, i.e., the point of light in front of the eye which has been considered above. The image of a real light behind the patient's head, formed by a concave mirror, is situated in front of the mirror. If the mirror is tilted up, the image moves up. The image of a real light behind the patient's head, formed by a plane mirror, is situated as far behind the mirror as the light is in front of it. When the mirror is tilted up, the image moves down.

Hence under the actual conditions of retinoscopy with a *plane mirror*, when the mirror is tilted to the right the immediate source of light moves to the left, and—

(a) in the hypermetropic eye, the circle of light on the fundus and the shadow seen in the pupil move to the right ;

(b) in the myopic eye (above — 1 D) the circle of light on the fundus moves to the right, and the shadow seen in the pupil moves to the left ;

(c) in the myopic eye of — 1 D there is no shadow ;

(d) in emmetropia and myopia of less than -1 D there is a very faint shadow moving to the right.

Stated as a mere guide to practice, with the plane mirror the shadow moves in the same direction as the mirror in hypermetropia and in the opposite direction in myopia above one dioptré; in myopia of one dioptré there is no shadow and in emmetropia and myopia of less than one dioptré there is a very faint shadow moving in the same direction as the mirror.

In actual retinoscopy the whole of the image of the illuminated area of fundus cannot be seen at once; the shadow is part of the circumference. In high degrees of ametropia the shadow has a distinctly curved border, it is very dark, and it moves slowly (Fig. 79). In low degrees of ametropia the border of the shadow looks straight; it is faint, and it moves rapidly.

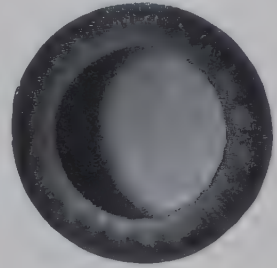


FIG. 79.

The movement of the shadow, being a purely optical phenomenon, is, of course, independent of the cause of the ametropia. Consequently, in astigmatism, if one axis is hypermetropic and the other myopic (mixed astigmatism) the shadow moves in opposite directions in the two meridians. Often the periphery of the cornea is flatter than the centre; correction of the refraction of the central part, which is the more important, will then differ from that of the peripheral part. These variations produce very puzzling shadows in many cases.

The Practice of Retinoscopy. Retinoscopy is conducted in a dark room at least six metres long. The surgeon sits at one metre from the patient. The patient wears a trial frame (Fig. 80)

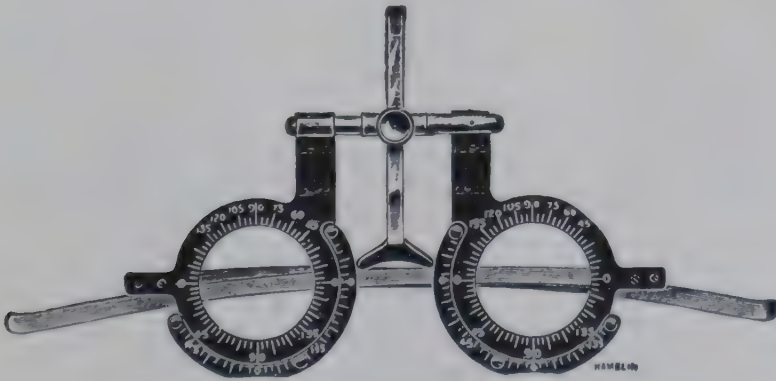


FIG. 80. Trial frame (Hamblin).

and fixes a spot of light at the far end of the room. A light may be placed behind and above the patient's head and the surgeon manipulates a plane mirror, perforated with a central hole through which he looks as he reflects light into the patient's eye, or he may use a

self-luminous retinoscope with a corresponding optical arrangement.

The light is reflected into the eye, and as the mirror is slowly tilted from one side to the other, the direction in which the shadow moves is noted. The horizontal meridian should be observed first, then the vertical. If the shadow appears to swirl round, not moving in the same meridian as the mirror, the eye is astigmatic, and the mirror is not moving in a direction which corresponds to either axis. A direction of movement can then be found in which the shadow will move either directly with or against the mirror; this is one of the principal axes of the astigmatism. The other axis is at right angles in regular astigmatism.

If the shadow moves with the mirror, progressively stronger convex lenses are put in the trial frame in front of the eye until no shadow can be seen. A still stronger convex glass is placed in the frame when the shadow probably moves against the mirror. We now know that the refraction has been over-corrected. The point at which there is absolutely no shadow—the point of reversal—is somewhere between the last two lenses, and we know that at that point the refraction of the eye *minus* the lens is equivalent to one diopetre of myopia.

If, for example, the shadow can still be seen to move with the mirror with $+4$ D lens in the frame, and moves against it with $+4.5$ D, we shall not be far wrong in considering that the point of reversal is $+4.25$ D. A lens of $+4.25$ D would therefore make the eye one diopetre myopic. The actual refraction is therefore $+3.25$ D.

Similarly, for spherical myopia, if -4 D eliminates the shadow against the mirror and -4.5 D gives a distinct shadow with the mirror, we know that -4.25 D will still leave the eye with -1 D. Hence the refraction is -5.25 D.

In astigmatism each principal meridian is corrected separately in the same way. When one meridian is approximately corrected the

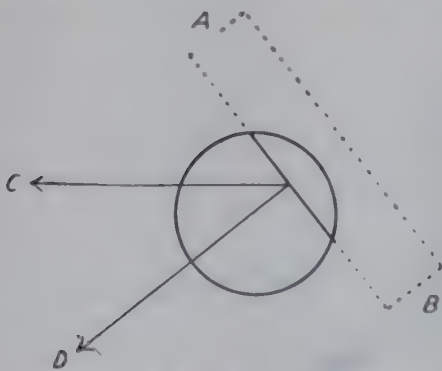


FIG. 81.

shadow assumes the shape of a band, the edge of the band being parallel to the axis of the corrected meridian. Even if the light is not moved in a direction accurately at right angles to this meridian, the shadow still seems to move in the same direction. This is due to an optical illusion. If, in Fig. 81, a straight edge, AB, is placed obliquely behind a circular hole in a card and is then moved horizontally in the direction of the

arrow C, it will appear to be moving in the direction of the arrow D at right angles to its own edge. The shadow is most sharply defined if the mirror is moved at right angles to its edge, i.e., at right angles to the corrected meridian.

The strength and direction of the axis of the cylinder are then verified by placing the appropriate sphere and cylinder in the trial frame and again studying the shadow effects. If there is any shadow in any direction the appropriate correction should be made. A further accurate test may be made by the surgeon leaning towards and then away from the patient, repeating his observations on each occasion. In the first case a shadow should move in the same direction as the tilt of the mirror, in the second in the opposite direction. If the expected change does not occur in both directions symmetrically, the correction is wrong.

Streak Retinoscopy, wherein, instead of a circular source of light as is obtained by an ordinary plane mirror, a streak of light is used, has some advantages. The streak effect is obtained by using a plano-cylindrical retinoscopy mirror or a similarly adjusted electric retinoscope. The appearances are more dramatic (Figs. 82 to 84). The

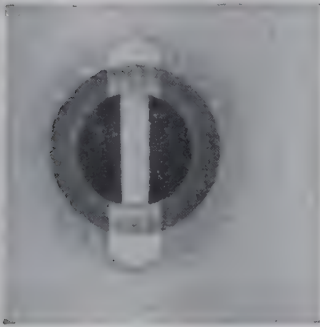


FIG. 82.

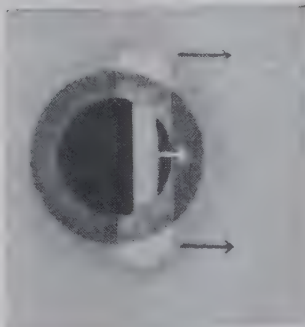


FIG. 83.

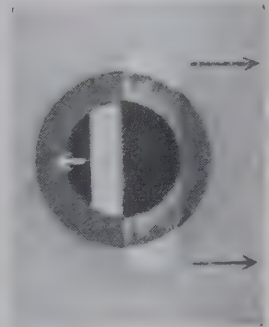


FIG. 84.

FIGS. 82-84. Streak retinoscopy. Fig. 82 shows the movements of the streak of light in the pupil with the reflection from the outer eye. Fig. 83, the neutralization point; the pupil is filled with light. Fig. 84, the appearance when the streak is not in the axis of astigmatism.

band of light in the pupillary aperture moves "with" or "against" the band of light outside the pupil, the axis of astigmatism is more easily determined, and on neutralization the streak disappears and the pupil appears completely light or completely dark.

Cycloplegics in Refraction. The use of cycloplegics, whereby the ciliary muscle is paralysed and the pupil dilated, has definite indications and contra-indications. Because of their strong accommodative reserve, very young people should always be given atropine or hyoscine, but less powerful drugs should be used with most hypermetropes below sixteen. In older patients the ideal refraction is the one estimated in the absence of cycloplegics. There is no need for cycloplegia as a routine although the pupillary dilatation is helpful for the beginner. They should be used, however, if there is a suspicion that the accommodation is abnormally active, if the

objective findings by retinoscopy do not agree with the patient's subjective desires, if definite symptoms of accommodative asthenopia are present which do not seem to be explicable by the error found without a cycloplegic, and if the pupil is small and the refraction presents technical difficulties. A mydriatic may also be indicated for ophthalmoscopic purposes, in order to see the macula or the periphery of the fundus. It is to be remembered, however, that the refraction under cycloplegia is pathological because the shape of the lens has been altered, and after the lens has assumed its normal shape, minute errors cannot reasonably be transposed to the dioptric system in the ordinary conditions of use; a post-cycloplegic test is therefore advisable. Moreover, with a mydriatic the refraction of the peripheral part of the lens is often estimated, not the central part which in practice is used for vision. When the refraction is estimated under cycloplegia a correction must be made to compensate for the normal tone of the ciliary muscle. As an average one dioptré is deducted, somewhat more in young hypermetropes and somewhat less in myopes.

Atropine is the most powerful cycloplegic and for young children should be instilled two or three times a day for three days before examination; in older children a drop of 0.05 per cent. hyoscine in oil is effective after an hour. Homatropine (2 per cent.) combined with cocaine (2 per cent.) is usually effective for adults if instilled two or three times, but a more rapid and transient effect is produced by such synthetic drugs as cyclopentolate hydrochloride (1 per cent.). If cycloplegia is desired, however, its effect, which varies greatly in different people and even in the two eyes of the same person, should be tested by estimating the residual accommodation which should not exceed one dioptré. Any mydriatic should be used with care in adults in whom the angle of the anterior chamber is narrow, owing to the danger of glaucoma. In older people mydriasis should be counteracted by pilocarpine (1 per cent.); and if suspicion of a tendency to closed-angle glaucoma exists and dilatation of the pupil is necessary, cocaine itself may be used.

To avoid ambiguity in ordering glasses the axes of cylinders should be uniformly numbered according to the method recommended by the International Council of Ophthalmology (Fig. 85).

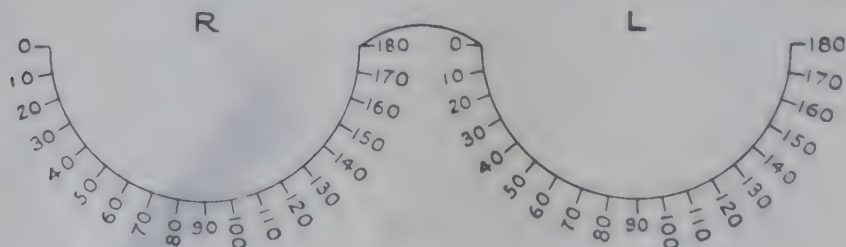


FIG. 85. The standard notation of axes of cylinders.

Difficulties in Retinoscopy. The shadows in regular astigmatism are not always easy to correct, owing chiefly to differences in curvature of different parts of the cornea. Usually the periphery of the cornea is flatter than the centre. The centre of the pupillary area will then be corrected by a different lens from the periphery, especially when the pupil is dilated. Various conflicting shadows may thus be seen, the commonest being the so-called "scissors" shadows, where two shadows appear to meet each other and cross as the light is moved in a given direction (Fig. 86). These difficulties are diminished with the undilated pupil. In irregular astigmatism the shadows move in various directions in different parts of the pupillary area and an accurate correction cannot be made by spherical or cylindrical lenses. In conical cornea a triangular shadow with its apex at the apex of the cone appears to swirl round its apex as the mirror is moved.

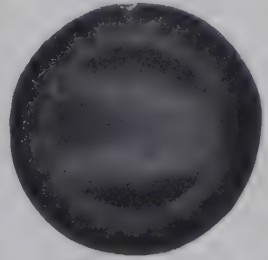


FIG. 86.

In conclusion, a word of warning must be given. The correction of a given refraction by retinoscopy may be easy or difficult. A large number of refractions should have been carefully corrected and confirmed by subjective tests before the beginner should consider himself justified in ordering spectacles without supervision.

Retinoscopy for Near Vision. The method of retinoscopy just described gives an objective measurement of the static refraction for distant vision. *Dynamic retinoscopy* has been introduced to give a similarly objective basis of the refraction of the eye when focused for near vision. The principle employed is to perform retinoscopy at the working distance with a self-luminous retinoscope on which is set a target for which the patient accommodates. The method gives an indication of the dynamic refraction, but our knowledge of the problems thus raised is insufficient to allow dogmatic conclusions to be drawn from the subtleties of the findings.

REFRACTOMETRY AND KERATOMETRY

Refractometry utilizes the principle of indirect ophthalmoscopy wherein a condensing lens brings rays emergent from the retina to a focus at a convenient distance. The rays from a test-object are collimated to enter the pupil as a parallel beam, and consequently, if the eye is emmetropic, are focused on the retina; emerging from the eye as a parallel beam, they are focused again by the objective lens at the position of the test-object. If the eye is myopic the emergent rays will be convergent and the image will be formed at a nearer point; if hypermetropic, the emergent rays will be divergent and the image will be formed further away by an amount depending on the degree of ametropia. This is estimated by the direct observation of this image, the end-point being the maximum sharpness of focus. A more accurate device is to employ the principle of displacement by parallax which was elaborated by Henker in his *parallax refractometer*. By displacing the line of view to one side, the optical system is arranged so that if (as in

emmetropia) the distances of the object and the image from the objective lens are equal, the image will be superimposed on the object and will not be seen. If (in myopia) the image is nearer the lens than the test-object, it will be displaced to the side next the illuminating tube. If (in hypermetropia) it is further away, it will be displaced to the other side. When the two do not coincide the test-object is moved until coincidence is attained when the refraction can be read from a scale.

A more accurate principle has been utilized by Fincham and Hartinger in their *coincidence optometers*. In these instruments, when the target is not in a position which is conjugate to the subject's retina, the retinal image is displaced from the axis. The image is viewed through a system of prisms which divides the field into two and reverses one half, so that when the image is out of alignment the halves of the line-image move in opposite directions; the setting is correct only when an unbroken line is formed.

The *keratometer* (*ophthalmometer*) measures the astigmatism of the anterior surface of the cornea at two points about 1.25 mm. on either side of its centre. Since considerable lenticular astigmatism may exist, the technique is untrustworthy except in aphakia. The method is based on the fact that the surface of the cornea acts as a convex mirror so that the size of the image reflected by it varies with the curvature: the greater the curvature of the mirror, the smaller the image. To measure the size of the image a device is employed, originally adopted by Thomas Young, of doubling the images by a double refracting prism. The object consists of two illuminated "mires" (AB, Fig. 87) disposed on a rotatable circular arc, and the curvature of any diameter of the cornea can be measured by observation through the telescope (T). The mires are shaped as in *ab* (Fig. 88) and are considered as the ends of a luminous object which appears in the cornea in duplicate as *ab* and *a'b'*. A and B are adjusted on the arc so that the two images *a'b'* just touch each other as in Fig. 88. The arc is now rotated through 90° and a similar reading made. If *a'* and *b* still touch there is no astigmatism. If the curvature in this

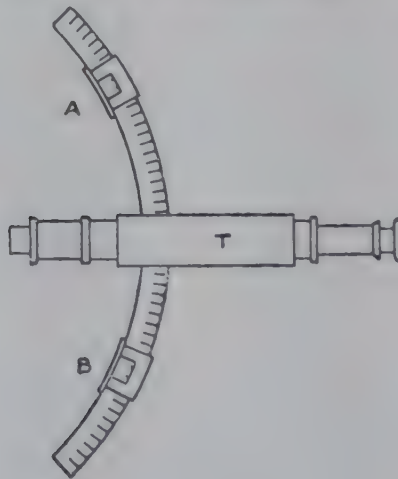


FIG. 87

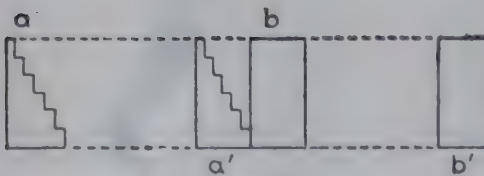


FIG. 88.

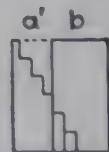


FIG. 89.

Figs. 87-89. The principle of the keratometer of Javal and Schiötz.

meridian is greater, the image is smaller and the mires will overlap as in Fig. 89. The mire a' is so constructed that each step corresponds to a diopetre of refractive power, the number of dioptries of astigmatism being thus read off directly.

THE SUBJECTIVE VERIFICATION OF THE REFRACTION

After the refraction has been estimated objectively it should always be verified subjectively by testing the visual acuity (p. 131), and if a cycloplegic has been used, the process should ideally be repeated in a *post-cycloplegic test*. These tests are made with the appropriate lenses, as found by the objective test, inserted in the trial frame (Fig. 80). Each eye is tested separately while an opaque disc is placed in the other compartment of the frame, and then the two are finally tested together.

If a cycloplegic has not been used the patient is asked to read the test types, and the effects of slight modifications in the lenses are tried in each eye separately, any small change being made which gives a marked improvement in visual acuity.

These manoeuvres are greatly facilitated by the use of a *cross-cylinder*, a mixed cylindrical combination of various strengths in which the spherical component is one half the (opposite) power of the cylindrical with the axes at right angles (Fig. 90). The most convenient form is a combination of a -0.25 D sphere with a $+0.5$ D cylinder. To check the strength of the cylinder in the optical correction, the



FIG. 90. Cross-cylinder.

cylindrical axis of the cross-cylinder is first placed in the same direction as the axis of the cylinder in the trial frame and then perpendicular to it. In the first position the cylindrical correction is enhanced by 0.5 D, in the second it is diminished by the same amount. If the visual acuity is unimproved in either of these positions, the cylinder in the trial frame is correct. If the visual acuity is improved, a corresponding change should be made in the correction unless it is especially contra-indicated; and the new combination verified by running through the cycle again.

To check the axis of the cylinder the principles of obliquely crossed cylinders are applied. A moderately strong cross-cylinder (± 0.5 or ± 1.0) is held before the eye so that each axis lies alternately 45° to either side of the axis of the trial cylinder. If visual improvement is attained by one or other alternative, the correcting cylinder is turned slightly in the direction of the axis of the cylinder of the same denomination in the cross-cylinder. The test is then repeated several times until the position of the trial cylinder is found at which rotation of the cross-cylinder gives no alteration in distinctness in either position.

It is not always easy for the patient to give definite answers with the use of the test types alone, especially in cases of small degrees of astigmatism. In these the results may be confirmed by the use of some type of *astigmatic fan* (Fig. 91). On looking at such a figure, if any of the lines are seen more clearly than the others, astigmatism must be present; if the vertical lines are clear, the diffusion ellipses on the retina must be vertical, that is, the horizontal meridian must be more nearly emmetropic than the vertical and *vice versa* (Fig. 92). A cylinder placed in front of the eye with its axis horizontal will therefore correct the vertical meridian, and when the correct glass is found all the lines will appear equally distinct.

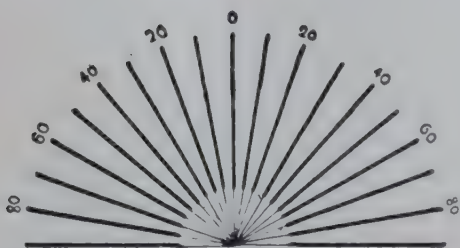


FIG. 91. Astigmatic fan.



FIG. 92. The optical effect of the astigmatic fan.

The cylinder which thus renders the outline of the whole fan equally clear is a measure of the amount of astigmatism, and the axis of the cylinder is at right angles to the line which was initially the most clearly defined.

As a clinical routine the test should be carried out with the patient's vision slightly fogged by an amount sufficient to over-correct every meridian by $+ 0.5$ D, and the patient is asked to observe if any of the lines stand out more clearly than the others. If astigmatism is present he will see one or a neighbouring group of lines more sharply defined by a degree depending on the amount of astigmatism; concave cylinders are now added, their axis lying at right angles to this until all the lines—including that at right angles to the first—are equally clear, additional convex spheres being added to maintain the fogging if necessary.

The entire examination must be done slowly and leisurely, and the patient is given the strongest hypermetropic or the weakest myopic correction with which he can attain normal vision.

The Correction of Near Vision should be preceded by the determination of the near point with the distance correction in place.

For this purpose appropriate test-types should be used. Snellen's reading test-types were constructed on the same principle as his distance

types (p. 131) and are therefore theoretically accurate. Ordinary types in common use, however, are more legible and more easily obtained. Jaeger, therefore, introduced a series of test-types in print such as was in common use a century ago corresponding so closely in size to those of Snellen as to be sufficiently accurate for practical purposes. These are still widely used and the sizes of print are numbered J1, J2, etc. Recently a similar card of modern types has been standardized by the Faculty of Ophthalmologists numbered from N5 to N48, corresponding to the modern Times Roman type in various sizes from 5 pt. to 48 pt.

The patient is given the reading test types and asked to hold them at the distance at which he is accustomed to work or read. When they are not distinctly seen, appropriate convex lenses should be added to the distance correction so that the near point is brought within the working distance, and the types are easily and comfortably read. The position of the *near point* should now be determined. This is most accurately done by approximating to the eye a card on which is drawn a fine line 0.2 mm. in breadth, until the line appears blurred (not doubled). For practical purposes it is sufficient to use the smallest test-type and move it towards the eye until it can no longer be easily read. The last position at which it can be read gives the near point. The distance of the near point from the eye is then measured with a tape. This distance is transformed, if necessary, into millimetres (25 mm. = 1 inch), and the range of accommodation is deduced from the formula $A = P - R$. The correction given should be such that some amplitude of accommodation (about one-third) is kept in reserve.

Presbyopic spectacles should never be prescribed mechanically by ordering an approximate addition varying with the age of the patient. Each patient should be tested individually, for the individual variation is large, and those lenses should be ordered in each case which give the most serviceable and comfortable, not necessarily the clearest, vision for the particular work for which the spectacles are intended. In all cases it is better to under-correct than to over-correct since, if the spectacles tend to be too strong, difficulties will be experienced with convergence, and the range of vision will be limited. In any case, lenses which bring the near point closer than 28 cm. are rarely well tolerated (that is, a total power of 3.5 D), and if for any reason the demands of fine work require a higher correction, the convergence should be aided with prisms as well as the accommodation with spheres (see Chapter 30).

THE CORRECTION OF ERRORS OF REFRACTION

The correction of errors of refraction has already been briefly sketched. It will be well, however, to outline the method to be adopted in systematically examining for and correcting these errors, and to indicate the requirements which should be satisfied by spectacles

1. External examination in diffuse light (Chapter 10).
2. Examination of the motility of the eyes (Chapter 29).
3. The cover test to elicit heterophoria and squint (Chapter 30).

This is best done at this stage. The detection of a squint may account for a marked deficiency of vision in the deviating eye, which, if it is not recognized early in the examination, may give rise to some concern.

4. The examination of the eyes by focal illumination, and by the plane mirror, and the ophthalmoscopic examination by the indirect and direct methods (Chapters 10–11).

5. The trial frames are put on and centred.

6. The testing of visual acuity, unilocularly and binocularly (Chapter 13).

7. The retinoscopy.

8. The subjective verification of the retinoscopy with the test types, astigmatic fan and cross-cylinders.

9. With the full correction in place, the testing of the muscular balance for distant vision.

10. With the full correction in place, the determination of the near point of accommodation and convergence (Chapter 28).

11. The addition of the correction for near work (if necessary), and the testing of the acuity with the near types, unilocularly and binocularly.

12. With the additional correction for near work, the estimation of the muscle balance for near vision (Chapter 30).

If the patient is less than five years of age (1) to (5) are done on the first visit. Then order ung. atropinæ, 1 per cent., to be inserted three times a day for three days. The ophthalmoscopic examination is repeated and (6), if possible, and (7) are done. The spectacles may then be ordered with the appropriate deduction for cycloplegia.

If the patient is between five and fifteen the same procedure should be undertaken but the entire examination can be done at one visit using cyclopentolate (1.0 per cent.) or 0.05 per cent. hyoscine in oil as a cycloplegic.

If the patient is between fifteen and twenty the same procedure should be undertaken, but the lowered activity of the ciliary muscle and the economic disability imposed by long cycloplegia allow cyclopentolate or homatropine with cocaine to be employed; a post-cycloplegic test is advisable.

If the patient is between twenty and forty cycloplegia followed by a post-cycloplegic test may be used as seems indicated (p. 74) but is usually not required. (1) to (10) should be performed as a routine.

If the patient is above forty cycloplegia is rarely necessary, should only be used with care and should always be neutralized by the instillation of 1.0 per cent. pilocarpine. Mydriasis (as with cocaine) may be employed in difficult cases. (1) to (12) should be performed as a routine.

Spectacles. In children, spectacles with large round or oval lenses should be ordered, otherwise the child may look over them. In adults with astigmatism rigid spectacles must be ordered.

It is very important that all spectacles fit accurately. For distant vision the lenses must be centred so that the optical centres are exactly opposite the centres of the pupils when the visual axes are parallel. For near vision the lenses are decentred slightly inwards and tilted so that the surfaces form an angle of 15° with the

plane of the face: they are then approximately at right angles to the visual axes when the eyes are directed downwards in reading.

Various forms of bifocal or trifocal lenses are sometimes used. In the former the upper part contains the distant correction, the lower part the near; in the second a strip for an intermediate distance is interposed between the two; in multifocal lenses a continuous gradation from the near to the far point is incorporated. If any of these are recommended, patients should be warned that they may experience some initial difficulty in moving about, particularly going downstairs, since vision through the reading portion of the lenses will be blurred and prismatic effects cause the apparent displacement of objects.

If tinted glasses are desirable, as in high myopia, albinism or in tropical countries, the correcting lenses may be tinted.

Contact Lenses. In cases of irregular corneal astigmatism and high myopia great improvement of vision occurs when a suitably curved glass meniscus is in actual apposition to the cornea or separated from it by a thin fluid meniscus. Contact lenses are made of plastic; if resting on the sclera they must fit with great accuracy, but micro-lenses resting on the cornea are easier to fit and to wear, although they sometimes may cause epithelial abrasions. As optical instruments they undoubtedly form the theoretically ideal correction for ametropia and are free of many of the disadvantages of spectacles. In all cases the prismatic effects of spectacles are eliminated and the field wherein clear vision is possible greatly increased; they are therefore particularly valuable in high errors of refraction, especially myopia or aphakia. Moreover, their effect in tending to maintain the size of the image approximately that of the emmetropic eye, makes them useful in cases of anisometropia, wherein the refractions of the two eyes are widely different (the most dramatic example of which is unilateral aphakia); while, since these lenses eliminate the corneal curvatures, high errors of astigmatism, as in conical cornea, are optically abolished. They cannot, however, be tolerated in all cases, their fitting must be of extreme accuracy, and perseverance is necessary to acquire facility in their insertion and removal. Even at the best they are not free from the danger of causing corneal injury unless they are used with care.

In cases wherein retinal disease is a cause of visual failure, **telescopic spectacles** may provide sufficient magnification to permit reading. They incorporate the optical principles of a Galilean telescope, but their use is difficult, particularly in view of the small size of the visual field. In such cases an ordinary convex lens held in the hand as a reading lens is often found more useful.

CHAPTER 8

ERRORS OF REFRACTION

Ametropia, that condition in which incident parallel rays of light do not come to a focus upon the light-sensitive layer of the retina, may be due to one or more of the following conditions :—

A. Abnormal length of the globe—too long in myopia, too short in hypermetropia—*axial ametropia*.

B. Abnormal curvature of the refracting surfaces of the cornea or lens—too strong a curvature in myopia, too weak in hypermetropia—*curvature ametropia*.

C. Abnormal refractive indices of the media—*index ametropia*. In index myopia the refractive index, either of the cornea, the aqueous or of the lens (produced either by a high index in the nucleus or a low in the cortex or both) is too high, and that of the vitreous may be too low. In index hypermetropia the opposite conditions are operative, and the error is high when the lens is absent.

D. Abnormal position of the lens—displacement forwards in myopia, backwards in hypermetropia.

Of all these factors the axial length of the globe is perhaps the most important.

Emmetropic eyes may differ in length by as much as 1 to 2 mm., and the radius of curvature of the cornea may vary from 7 to 8 mm. Emme-

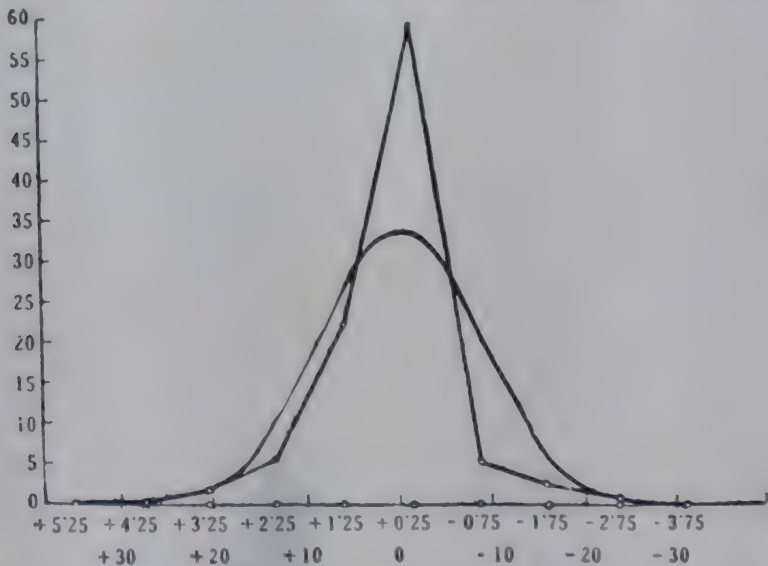


FIG. 93. The relative incidence of refractive errors. The refraction curves of Scheerer and Betsch (the higher curve) compared with the theoretically derived binomial variation curve (the lower curve and lower figures). The abscissæ are refractions measured without cycloplegia.

tropia therefore results from the integration of all the variables mentioned in the previous paragraph and a deviation in one factor is often compensated by the opposite tendency in another. Statistically one might expect its incidence to resemble the Gaussian frequency curve, but since the full development of emmetropia is never present normally at birth the curve will have a certain "skew deviation." Most infants are born hypermetropic; almost inevitably some cases will fail to reach emmetropia and remain hypermetropic, while others will proceed too far and become myopic. Of these the former are by far the more numerous. Most cases of low ametropia (including myopia) are merely biological variants around a mean and cannot be regarded as pathological (Fig. 93).

Myopia, or "short sight," is that dioptric condition of the eye in which, with the accommodation at rest, incident parallel rays come to a focus anterior to the light-sensitive layer of the retina. The majority of cases merely result as variants in the frequency curve of axial length and curvature, the former being the more important although curvature myopia occurs commonly as a factor in astigmatism. Such cases of *simple myopia* are in no sense pathological, there are no degenerative changes in the fundus although peripheral retinal degeneration often becomes evident in later life, and they do not progress after adolescence when a degree of 5 or 6 dioptres may be attained. In severe illness, however, or states of debility, the sclera may stretch and the myopia increase.

Rarely a *developmental myopia* occurs. In this case the child is born with an abnormally long eye, the fundus may lack pigmentation and the choroidal vessels are evident while a myopic crescent (p. 254) may be seen at the disc. The refraction soon after birth may be — 10 D; but in this type of case progression is rare.

Pathological axial myopia is degenerative and progressive. The refractive change appears in childhood, usually between the ages of five and ten, and increases steadily up to twenty-five or beyond, finally amounting to 15 to 25 D or more. The degenerative changes in the fundus, on the other hand, do not appear until later in life, becoming marked at about the fifth decade. The condition is strongly hereditary, being commoner in women than in men. It has racial tendencies, being common, for example, among Jews and Japanese, and most cases are of genetic origin. Many other ætiological theories have been advanced—excessive accommodation and convergence in near work, vascular congestion due to a dependent position of the head, and so on—but they have little to recommend them.

The condition is essentially a disturbance of growth on which are imposed the degenerative phenomena; these will be considered at a later stage (p. 254). Endocrine or nutritional disturbances, debility or illness, probably act as incidental factors which may increase the general tendency; but, despite popular belief which

still lingers, environmental conditions such as excessive near work probably have little influence upon the condition which is genetically predetermined, except in so far as they inhibit normal healthy development.

Pathological *curvature myopia* is seen typically in conical cornea. *Index myopia* accounts for myopia as a premonitory symptom of senile cataract, when it is due to the increased refractive index of the nucleus of the lens; it also accounts for myopia in some cases of diabetes, with or without cataractous changes in the lens.

In degenerative axial myopia the increase in length of the eye affects the posterior pole and the surrounding area; the part of the eye anterior to the equator may be normal (Fig. 94). The elongation

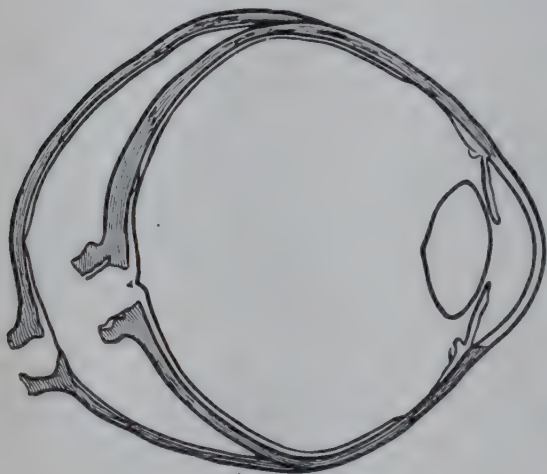


FIG. 94. The two eyes of the same individual superimposed: the one emmetropic, the other with -15 D of myopia.

is probably not due to stretching but to a primary degeneration of the coats of the eye including the posterior half of the sclera. In the high degrees, the sclera may bulge out at the posterior pole to form a *posterior staphyloma*, distinguishable clinically by the optical condition and the associated changes in the fundus. The edges of the bulge may be actually visible by the indirect method of ophthalmoscopy owing to the presence of a crescentic shadow two or three disc-diameters to the temporal side of the disc and concentric with it and to the change in course of the retinal vessels.

Two typical ophthalmoscopic appearances are seen in high myopia—changes at the disc typified in the development of a myopic crescent, and changes in the central area of the fundus described as chorio-retinal myopic degeneration (p. 254). Degenerative changes also occur at the periphery of the retina which may lead to the occurrence of a retinal detachment, and degenerative changes in the vitreous are common, giving rise to dust-like vitreous opacities or large floaters composed of elements of the vitreous framework. These “floaters” are seen more plainly by myopic than by other eyes because the entoptic image is larger. These degenerative changes may have serious visual

consequences ; in fact they are among the more common causes of severe visual disability.

The only symptom in low myopia may be indistinct distant vision. In other cases and in high myopia there is often, in addition, discomfort after near work, due largely to disproportion between the efforts of accommodation and convergence (*q.v.*). The eyes may be sensitive to light. Black spots may be seen floating before them, and sometimes flashes of light are noticed ; the latter may occur irrespective of any tendency to detachment of the retina. In very high myopia the eyes are prominent, the pupils are large, and the anterior chamber appears deeper than normal, probably only owing to the dilatation of the pupil. There may be an apparent convergent squint due to a large negative angle γ (*q.v.*). Vision may be very poor, even with optical correction ; scotomata may be present, both central and peripheral.

As regards *prognosis*, low or moderate degrees of simple myopia (up to 5 or 6 D), unless occurring in young children, have a good prognosis. They are not likely to progress, and in some of the conditions of civilized life they may even be an advantage to the individual. The same condition in a child before the age of six or seven should give rise to anxiety if it is not of the congenital type, since the degenerative condition is clinically indistinguishable from the simple at this stage. The former is of grave prognosis, because it is almost certain to progress so that eventually there may be 10 or 15 D of myopia or more, accompanied by serious degenerative changes in the fundus and defects of vision. The likelihood of these developments must be judged by the acuity of vision after correction, the condition of the fundus and the evidence of heredity.

Treatment consists in wearing suitable correcting spectacles and attention to the hygiene of the eyes. Each case must be considered on its merits.

With regard to the ordering of spectacles in myopia, every surgeon agrees that *myopia must never be over-corrected*. Opinions differ as to details. In low myopia, up to 5 or 6 D, no harm is done by ordering the full distance correction for constant use, and if this is done the patient must be warned not to hold near work closer than ordinary reading distance. Many surgeons order lenses weaker by 2 or 3 D for near work with a view to diminishing accommodation. As we have seen, this is based on an old and erroneous ætiological view, but many patients are more comfortable for near work with the weaker lenses. Any advantage in comfort in the young, however, tends to be neutralized by the disturbance of the normal relationship between accommodation and convergence.

In practice, in low myopia, the full correction may be ordered for constant use ; in the event of any discomfort being experienced, weaker lenses may be ordered for near work, especially if much reading or close work is done. Children should wear their distance

correction constantly—not particularly in the interests of their eyes but in the interests of their mental development—for children with even low degrees of uncorrected myopia cannot be expected to take a normal interest in their surroundings since they cannot see distant objects as clearly as their fellows. Their mental horizon is constricted, they tend to become unduly introspective, and they are thrown more and more into finding their interest in reading and near work. Adults need not wear their correction constantly in the absence of symptoms provided they resign themselves, when they do not wear spectacles, to their poor vision and do not impose the strain upon their eyes of attempting to see the difficult or impossible. In low degrees of error, spectacles for near work are rarely required after the presbyopic age.

In high myopia it is wise always slightly to under-correct even for distance, and the same or still weaker lenses may be ordered for near work. In the highest grades the patient often sees best with lenses which are decidedly weaker than the full correction; he should be allowed to choose those he prefers. One reason is that strong minus lenses considerably diminish the size of the retinal images and make them very bright and clear. The retinal images are diminished because the lenses have to be worn farther from the eye than the anterior focal plane (p. 59); spectacles for high myopia should therefore be made to fit as closely to the eyes as possible; toric lenses may be ordered, or the eyelashes cut in order to prevent them from rubbing upon the lens. The very bright, clear images are uncomfortable because the retina has become accustomed to large and indistinct images. Moreover, much artificial astigmatism and therefore distortion of the image, is produced by looking obliquely through strong lenses. Very short-sighted people thus get into the habit of turning the head rather than the eyes to avoid looking obliquely through the lenses. Indeed, some high myopes can find their way about better without any spectacles. Contact lenses may be of great value in cases of this type when they can be tolerated.

In very high myopia the requisite amount of convergence for near work, if spectacles are not constantly worn, may be impossible. In this case the effort to converge is abandoned so that reading and other near work become unocular and the disused eye becomes divergent.

As regards hygienic measures in myopia, especially in the young, near work, apart from being held in the proper position and being undertaken in good illumination, need not be restricted if the general health and physical development of the child are not being undermined thereby. Only if the visual acuity of the child is such as to make it difficult for him to keep pace with his fellows at school need special educational methods be adopted wherein most of the teaching is oral and visual instruction is limited to specially printed large types. In all cases the most important factor is the maintenance

of the general health, and if the myopia is rapidly progressive, and especially if the child is under stress, the temporary cessation of schooling and a change of air to the country with plenty of healthy exercise may well be desirable from this point of view.

Owing to the gravity of the prognosis in later life, for economic reasons high myopes with degenerative changes in the fundus or a family history thereof should avoid an occupation wherein close work is necessary. Consideration should also be given to the hereditary propagation of the disease ; at the least, two high myopes with pronounced degenerative changes in the fundi should not have children.

Operative treatment for high myopia. If an eye has axial myopia of 21 D, its length will be about 31 mm. (p. 90). If the crystalline lens of such an eye is removed, parallel rays will be focused upon the retina without the intervention of any correcting lens, and the retinal images of distant objects will be larger than those of the emmetropic eye. Hence extraction of the lens has been advocated in high myopia often with immediately satisfactory results. The operation is, however, attended with considerable danger for such eyes withstand operative measures badly, the vitreous is likely to be fluid and the retina and choroid are probably degenerate so that the tendency to retinal detachment is increased.

Hypermetropia (*Hyperopia*), or "far sight," is that dioptric condition of the eye in which, with the accommodation at rest, incident parallel rays come to a focus posterior to the light-sensitive layer of the retina.

As in myopia, the chief factor in clinical hypermetropia is *axial*—an abnormal shortness in the length of the eye. It must be remembered that a small eye, although too short, is not necessarily hypermetropic since there may be uniform diminution of all the parts. This is, perhaps, most easily understood if a diagram such as Fig. 56 is considered ; if such a diagram is uniformly diminished, as by photography, the parallel rays will still come to a focus on the retina. As a matter of fact, however, highly hypermetropic eyes are almost invariably also smaller than normal.

Curvature hypermetropia occurs commonly as a factor in astigmatism ; it is almost unknown as a cause of spherical hypermetropia. *Index hypermetropia* accounts for the hypermetropia of old age (p. 66), and it is to be attributed to the increased refractive index of the cortex of the lens.

Hypermetropia rarely exceeds 6 to 7 D, which is equivalent to a shortening of the optic axis of 2 mm. Individual cases of much higher degrees without other anomaly have been recorded—up to 24 D.

In the young the condition may cause no symptoms. When symptoms are present or arise, they are chiefly referable to the abnormal amount of accommodation to which these eyes are sub-

jected, and to the lack of balance between accommodation and convergence (*q.v.*). As has been pointed out, the healthy youth has an ample reserve of accommodation, and if he happens to be hypermetropic he accommodates for distant and near objects without being conscious of the act. If he is weakly or does much near work the perpetual overaction of the ciliary muscle is likely to produce symptoms ; the condition is often called *accommodative asthenopia* or "eye-strain." The symptoms are noticed chiefly after close work, especially in the evening by artificial illumination. The eyes ache and burn ; they may feel dry, so that blinking movements are more frequent than usual, or there may be lachrymation. The conjunctiva and edges of the lids become hyperæmic and if near work is persisted in, headaches, usually frontal, develop.

In young children hypermetropia is a predisposing cause of convergent strabismus (*q.v.*). Latent convergence is often found in hypermetropes, although other forms of heterophoria may occur (*q.v.*). The presence of heterophoria increases the tendency to headache and other symptoms of eye-strain.

In older patients no symptoms may be caused until the power of accommodation has diminished to the extent that the near point is beyond the range of comfortable reading distance and work has to be held farther off than usual in order to be seen clearly. The greater the degree of hypermetropia the sooner will this symptom arise ; in other words, apparent presbyopia commences at an earlier age than usual.

Ophthalmoscopically the fundus may exhibit no abnormality. A bright reflex, suggesting the appearance of watered silk, is commoner in hypermetropic than in emmetropic or myopic eyes ; and in some cases optic neuritis is nearly simulated—pseudo-papillitis (*q.v.*).

Anatomically, the smallness of the eye is not confined to the post-equatorial segment as in myopia, nor are abnormalities found in the retina or choroid. The diameter of the cornea is often reduced and regular astigmatism is common while the anterior chamber is shallower than usual, owing partly to the normal size of the lens, a configuration which predisposes to closed-angle glaucoma.

The new-born are almost invariably hypermetropic (average 2.5 D). In the first decades of life the incidence of hypermetropia falls rapidly, remaining at about 50 per cent. after the twentieth year. There is no predilection for either sex. It is interesting that primitive races and the higher mammals, especially the carnivora, are generally hypermetropic.

Treatment consists in prescribing the correcting lenses. Unless there are definite symptoms there is no reason for insisting upon the use of spectacles in the young or middle-aged. In elderly people the hypermetropia must be corrected for near work : the ordinary presbyopic addition must be added to the hypermetropic correction,

but care should be taken that these cases are rather under- than over-corrected.

Astigmatism is that condition of refraction in which a point of light cannot be made to produce a punctate image upon the retina by any spherical correcting lens. The varieties of regular astigmatism have been already enumerated (p. 58).

Regular astigmatism, the only form susceptible to optical correction by lenses, invariably produces some defect in visual acuity. It is particularly liable to cause the worst forms of asthenopia or "eye-strain"; the asthenopia in these cases is only in part accommodative. It is often worse in the lower degrees of astigmatism than in the higher because of endeavours to accommodate so as to produce a circle of least diffusion upon the retina (p. 58). Aching of the eyes and headaches are common symptoms; the eyes quickly become fatigued with reading and the letters are described as "running together."

Regular astigmatism is usually a congenital defect, due in most part to differences in the curvature of the cornea in different meridians. It must be remembered that frequently the cornea is not alone at fault, for corneal astigmatism may be increased or partially corrected by lenticular astigmatism. Regular astigmatism may be traumatic following a wound, frequently surgical, in the corneo-scleral margin since the contraction of the scar causes flattening of the cornea in the meridian at right angles to the wound. The astigmatism due to this cause continues to alter for some weeks after the injury so that final spectacles should not be ordered for at least six weeks thereafter.

The higher degrees of astigmatism cause much lowering of visual acuity: this is usually least in mixed astigmatism, probably because the circle of least diffusion falls upon or near the retina.

Treatment. If the astigmatic error is small and not associated with symptoms, spectacles are unnecessary unless the highest visual acuity is desired; but in all cases in which astigmatism causes asthenopic symptoms the full optical correction should be ordered for constant use, that is, both for distant and near vision.

Aphakia is the condition of the eye when the crystalline lens has been removed. The eye is extremely hypermetropic if it had been emmetropic or had only a low grade of ametropia before removal of the lens, and all accommodation is lost. The hypermetropia, as estimated by the correcting lens required when worn in the usual position, is about 10 or 11 D if the eye were previously emmetropic.

The optical conditions of the aphakic eye are very simple. It consists of a curved surface, the cornea, separating two media of different refractive indices, air and aqueous plus vitreous. Knowing the radius of curvature (8 mm.) and the refractive indices (1 and 1.33), it is easy to

calculate the focal distances ; the anterior focal distance is 23 mm. and the posterior 31 mm., as compared with 15 mm. and 24 mm. respectively for the normal eye. If the aphakic eye were 31 mm. long, parallel rays falling on the cornea would be brought to a focus on the retina and no correcting glass would be required for distance. The axial myopia of a phakic eye which is 31 mm. long equals -21 D.

The retinal image of the aphakic eye is about a quarter larger than the emmetropic retinal image. Hence vision of 6/6 with a correcting lens after extraction is not quite so good as it seems ; and, owing to the disparity of the images, any attempt to correct unilateral aphakia with spectacles when there is good vision in the other eye leads to an intolerable diplopia. With contact lenses, however, comfortable binocular vision may be attained.

In addition to the hypermetropia, there is always some astigmatism in those cases in which a corneal or corneo-scleral section has been made. If the section is in the upper part of the cornea, the astigmatism is against the rule since the cornea is flattened in the vertical meridian. The astigmatism usually amounts initially to 2 or 3 D but gradually diminishes.

Treatment. The refractive error is determined by retinoscopy and by subjective tests ; the ophthalmometer may afford help in determining astigmatism. The optical condition of aphakia with a strong correcting lens and with no accommodation is difficult and great patience is often necessary if the patient is to adapt himself to it. Sometimes these difficulties can be much improved by a contact lens provided the patient can manipulate and tolerate it.

Anisometropia is the condition in which the refractions of the two eyes show a considerable difference. A slight difference is very common but all varieties and degrees of anisometropia occur. The condition may cause asthenopic symptoms. In the lower grades there is usually binocular vision, although it is imperfect and the effort of fusion may produce symptoms of eye-strain. In the higher grades this is impossible ; vision is then unocular, and there is some danger of the eye which is not used becoming divergent.

Treatment. The correction of anisometropia is often difficult. It has already been mentioned that if correcting lenses are placed at the anterior focal plane of the eye, the retinal images in axial ametropia are the same size as the emmetropic retinal image. In practice the lenses are nearer to the eyes so that with convex lenses the retinal image is diminished, with concave, enlarged. In high grades of anisometropia, therefore, there will be a considerable difference in the size of the retinal images of the two eyes (*aniseikonia*). Patients find it difficult or impossible to fuse these sharp but diverse images. Moreover, on looking obliquely through the lenses the prismatic effect and the distortion are different in the two eyes, enhancing the discomfort. The use of ordinary spectacle lenses thus presents difficulties, and if a full correction cannot be

borne a compromise may be adopted wherein each eye is under-corrected. *Iseikonic* or *size-lenses*, which correct such a difference in their optical construction, require specialized methods of manufacture and their clinical results are often disappointing. Contact lenses diminish these optical defects and may be ordered in suitable cases. Alternatively, resort may be had to uniocular vision, one eye being used at a time, a habit which grows more comfortable with practice as is seen in the use of a uniocular microscope or monocle. It is particularly fortunate when one eye is nearly emmetropic and the other myopic for the former can be used for distant, the latter for near vision without the aid of spectacles.

CHAPTER 9

ANOMALIES OF ACCOMMODATION

Insufficiency of Accommodation. In this condition the accommodative power is below the lower limit of what might be accepted as normal for the patient's age ; in a sense presbyopia (p. 67) is a physiological failure of accommodation due to hardening of the lens. Such an insufficiency is usually due to weakness of the ciliary muscle, the ætiology embracing all the causes of muscular fatigue (general debility, anæmia, toxæmia, etc.) accompanied by excessive use of the eyes particularly for close work. A rapid failure of accommodation also occurs in the prodromal stages of glaucoma, due probably to impairment of the effectivity of the ciliary muscle by the increased pressure.

The symptoms are those of eye-strain with particular difficulty associated with near work. The treatment should be directed essentially to the causal condition, but if close work is difficult reading spectacles may be prescribed, the same procedure being adopted as that recommended for presbyopia. In general the weakest convex lenses which will allow adequate vision should be ordered so that the accommodation may be exercised and stimulated rather than relieved.

Paralysis of Accommodation, or *cycloplegia*, occurs in disease as well as from the direct action of cycloplegic drugs. Unilateral cycloplegia is generally due to drugs (often through rubbing the eyes after using a belladonna liniment), contusion (*q.v.*), or to paralysis of the third nerve. Bilateral paresis, less commonly paralysis, occurs typically after diphtheria, but may appear after debilitating illness, or with syphilis, diabetes, alcoholism and cerebral or meningeal diseases.

In diphtheritic cases the paralysis of accommodation follows the primary attack after an interval of several weeks, and is often associated with paralysis of the palate, loss of knee jerks, etc. The sore throat may have been very slight and its diphtheritic character unrecognized. The lesion is probably nuclear and toxic.

In complete paralysis the sphincter pupillæ is also generally paralysed so that the pupil is widely dilated. In paresis the pupil may be scarcely affected, especially after diphtheria, but in this disease the reverse of the Argyll Robertson pupil may be met with—loss of reaction to accommodation with retained reaction to light. The symptoms depend upon the condition of the refraction. If the patient is myopic, the defect may pass unnoticed ; if he is

emmetropic, near vision alone will be affected; if he is hypermetropic, both distant and near vision will be affected, but particularly the latter. In paresis it may be possible to diagnose the condition only by carefully measuring the range of accommodation.

The prognosis is good in cases due to drugs or diphtheria. In traumatic cases the condition may be permanent.

Treatment is that of the cause. Whenever the condition is bilateral, near work can be carried on by using suitable convex lenses, as in the correction of presbyopia. Miotics are sometimes used, but they may do harm and seldom do good.

Spasm of Accommodation. It has already been mentioned that the ciliary muscle has physiological tone which is abolished by atropine, and is equivalent to about one dioptré. In spasm of the ciliary muscle it is found that atropine produces a much greater effect. The condition is found only in young patients and, contrary to what might be expected, particularly in myopes. An actual or relative myopia is produced and in these cases subjective testing without a cycloplegic indicates too high an error. Spasm of accommodation is produced artificially by the instillation of miotics.

In spontaneous spasm of accommodation there is nearly always some error of refraction and the eyes have usually been subjected to too much near work in unfavourable circumstances which may include such factors as bad illumination, a bad position, mental stress and anxiety, and so on. The condition should not be diagnosed unless proved to be present by the use of atropine.

Treatment consists in the use of atropine for several weeks. The amount of near work must be limited and carried out under good conditions, the error of refraction being carefully corrected.

SECTION III

THE EXAMINATION OF THE EYE: THERAPEUTICS

CHAPTER 10

EXTERNAL EXAMINATION

In this section we shall confine ourselves essentially to conditions affecting the globe itself.

When the cornea is inflamed or ulcerated the eye is irritable and resistant to examination in bright light so that the slightest attempt to separate the lids is accompanied by violent blepharospasm, especially in children. When difficulty is encountered, any roughness, or even an amount of pressure which is quite justifiable in other cases, may suffice to cause perforation of an ulcer. In such a case the lids must be separated by retractors (Fig. 95).



FIG. 95. Lid retractor (half size).

With babies a special arrangement of the patient facilitates examination. The surgeon sits facing a nurse who holds the child on her lap, the baby's head being placed between the surgeon's knees. The nurse holds the child's hands against its body, thus keeping them out of the way, and at the same time steadying the child. Older children are placed upon a couch. The ends of the retractors are inserted gently between the lids and the globe and traction is then made not only in opposite directions up and down, but also away from the globe, so that the lids are lifted off the globe at the same time that they are separated. In these cases the globe tends to roll forcibly upwards when light falls upon it, which makes it difficult to see the eye even with the use of retractors. The difficulty may be overcome by pressing the end of the lower retractor well into the lower fornix, which drags the eye downwards by pulling on the bulbar conjunctiva.

If there is no fear of perforation we may dispense with retractors. In children an attempt to separate the lids when there is much blepharospasm is usually followed by eversion of both lids, the cornea still remaining hidden. The way to overcome this difficulty is to place the two thumbs close to the edges of the lids and to press gently but firmly upon the globe as the lids are drawn apart. In this manner they are separated without becoming everted, but we must be extremely careful not to exert undue pressure and not to touch the cornea with the thumb-nails.

Examination of the anterior segment of the eye is made by three methods : (1) General inspection in a good diffuse light. (2) Examination in focal (or oblique) illumination using the loupe or slit-

lamp. (3) Examination of the recess of the angle of the anterior chamber with the gonioscope. At this stage it will be convenient to discuss the two main clinical methods of focal illumination ; we shall describe gonioscopy separately.

Focal or oblique illumination using the loupe is carried out as follows : the patient is placed preferably in the darkroom, with a light about two feet in front but slightly to one side. The light is concentrated upon the cornea by a strong convex lens and the position of the minute image of the light thus formed can be moved over the surface of the cornea by slight lateral movements of the lens without altering the position of the light. Similarly the light may be focused upon the iris or crystalline lens by moving the lens slightly nearer to the eye. A small electric torch, the beam of which can be focused to a point or converted into a slit, is a more convenient source of illumination.

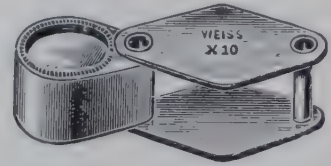


FIG. 96. Ophthalmic loupe.

Having thus brilliantly illuminated the area which we wish to investigate, we may magnify the spot by looking through a strong convex lens or corneal loupe held in the other hand (Fig. 96). The management of the two lenses requires a little practice, but is easily mastered. A few words of explanation about the corneal loupe will help us to employ it to best advantage.

When we magnify a small object with a strong convex lens we place it within the focal distance of the lens and view it through the lens. We know that under these conditions the lens forms an enlarged image upon the same side as the object, but farther away (Fig. 97). In order to see the image to the best advantage we must see as much of it as possible, and we must see it with the largest possible visual angle. The first requisite demands that the observer's eye shall be close to the lens. The second depends upon the relative

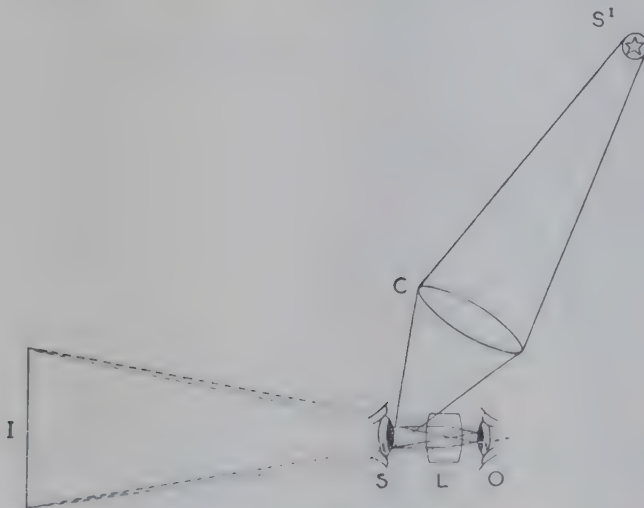


FIG. 97. The optical principles of oblique illumination. The light from a source (S') is concentrated by a lens (C) on the subject's eye (S) ; the observer (O), looking through the loupe (L), sees the magnified image (I).

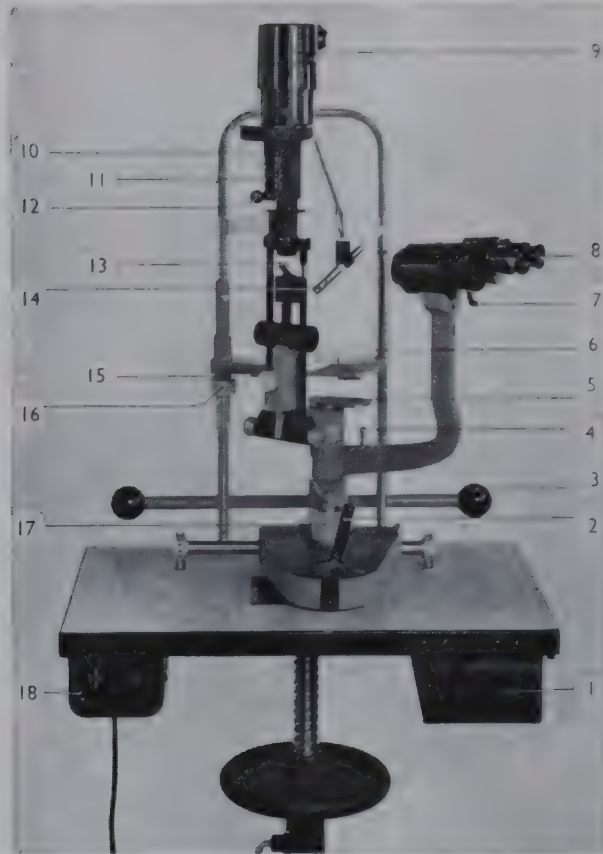


FIG. 98. The Haag-Streit Slit-lamp 900.

1. Accessory box.
2. Joystick lever for horizontal coarse and fine adjustments.
3. Gripping handles for patient.
4. Roll for setting the angle between microscope and illumination unit,
5. Guide-plate for pre-set lenses and applanation tonometer.
6. Chin-support.
7. Lever for changing objectives.
8. Interchangeable eye-pieces.
9. Lamp-casing.
10. Lever for four different light filters.
11. Lever for six different diaphragms.
12. Ball-handle for turning slit-image.
13. Interchangeable illumination mirror.
14. Fixation lamp with annular fixation marker.
15. Centring screw.
16. Level adjustment control for chin-support
17. Height adjustment control of slit-lamp.
18. Transformer with switch.

distances of the object and the eye from the lens ; in practice these are found by slight movements of the lens.

In employing focal illumination, first focus the light upon the required spot, then place the corneal loupe near the spot and look through it. Slowly advance the loupe towards the cornea until the spot comes into focus, then get the eye as close to the loupe as possible.

By moving the light and the loupe slowly over the whole surface of the cornea we can thoroughly explore it. By advancing the convex lens we can illuminate successively the back of the cornea, the iris and anterior part of the lens, and finally the deeper parts of the lens. By simultaneously advancing the position of the loupe towards the cornea we can successively bring these structures into accurate focus ; but we cannot see beyond the posterior region of the lens using a high-power loupe, as the focal distance is too short. Moreover, in order to examine the deeper parts of the lens, we must have the light almost in front of the patient.

With a *binocular loupe* a stereoscopic effect is obtained, and the depth of opacities can be determined with great accuracy, but the degree of magnification is less ; the greatest accuracy is attained with the slit-lamp.

The *slit-lamp*, introduced by Gullstrand, is essential when minute examination of the eye is required (Fig. 98). It employs the same principles of focal illumination wherein a brilliant light is brought to a focus as a slit or a point by an optical system supported on a movable arm, and observations are made through a binocular microscope. Fig. 99 shows a general view of the eye illuminated by a beam of light

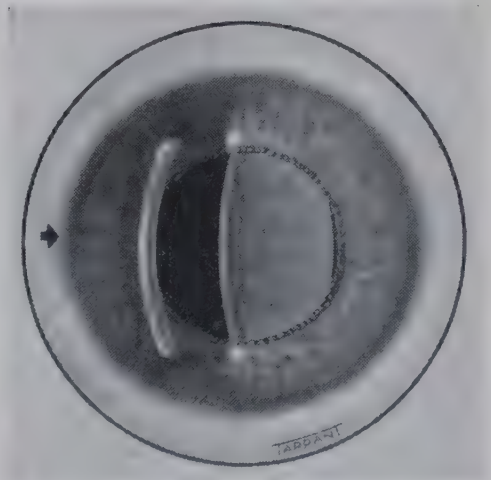


FIG. 99. Optical section of the normal eye. As seen with the slit-lamp. The light (arrowed) comes from the left and in the beam of the slit-lamp the sections of the cornea and the lens are clearly evident.

of moderate width coming from the slit-lamp entering the eye from the left side. Optically homogeneous media appear quite black ; structures like the cornea, lens, and suspended particles in the aqueous scatter the light. Hence, on the left of the diagram is seen the illuminated portion of the cornea forming a parallelepiped, the brighter area corresponding to the surface, the darker to the section of the cornea. The black space

to the right is the anterior chamber. Then follows the "phantom" of the lens, in which can be distinguished the dim central interval, formed by the embryonic nucleus with its Y-sutures, outside which are the successive "zones of discontinuity"—the foetal nucleus, the infantile nucleus, the adult nucleus, and the cortex. Still farther to the right is the faintly striated vitreous.

We shall see that a simple addition to the optical system makes possible a similar view of large areas of the posterior part of the fundus.

The Conjunctiva. In order to examine the whole conjunctival sac it is necessary to expose the palpebral conjunctiva and the fornices. The lower fornix is easily exposed by drawing down the lower lid while the patient looks towards the ceiling. The upper palpebral conjunctiva is exposed by everting the upper lid.

Eversion of the upper lid requires some practice. (1) The best, and often the easiest, method is as follows: Stand facing the patient. To evert the left upper lid, place the right thumb on the lower lid near the margin while the patient looks up. Then ask the patient to look down so that the upper lid tends to fall over the lower and the surgeon's thumb. Lay the side of the index finger along the upper lid above the tarsus exerting a steadying pressure upon it downwards. With the patient still looking down an upward rotatory movement of the thumb then everts the lid and as the finger is withdrawn the thumb is rotated to support the everted lid against the orbital margin. The right lid is everted in the same manner, using the left hand.

This method is very easy when the eyes are prominent, and it causes a minimum of discomfort to the patient. When the eyes are deeply set in the orbit, as is often the case in old people whose orbital fat has become to a great extent absorbed, more pressure is needed and a little discomfort is caused. In such cases the following method may be adopted: the tyro will generally find it easier:

(2) Place a probe or thin pencil horizontally along the skin of the upper lid at the level of the upper border of the tarsus, the patient looking towards his feet. Grasp the eyelashes between the left index and thumb, and draw the lid away from the globe, using the probe as a fixed point. Rotate the lid in a vertical direction round the probe, which is then withdrawn.

In many cases we wish to evert the upper lid when standing behind the patient, who may be lying on a couch. In this case the following is the best method:

(3) Place the left index finger vertically upon the lid while the patient is looking towards his feet. Grasp the lashes with the right index and thumb, and rotate the lid around the tip of the left index.

With babies the help of a nurse is necessary and the position described on p. 94 should be adopted. If, as is often the case, there is blepharospasm, eversion of the lids is extremely easy; however, it becomes troublesome when we wish to examine the cornea. Here the spasm of the orbicularis fixes the lids against the globe, and the slightest attempt to draw the lids apart causes both to become everted. When this does not occur, method (3) must be adopted.

Having everted the upper lid we can examine the palpebral conjunctiva, but we are still unable to see the upper fornix. This can usually be effected in adults by the following manœuvre:

(1) With the lid still everted by the first method, it is fixed in that position by placing the left thumb upon its margin at about the middle. The right

thumb or finger is placed in the middle of the lower lid. Firm, steady pressure is then made through the lower lid upon the globe in a direction straight backwards, as if to push the globe into the orbit. In the meantime firm pressure is also exerted backwards upon the upper lid with the left thumb. The fornix will generally start forwards suddenly, but only if the patient keeps looking well down towards his feet all the time.

This method, though unpleasant, is not painful. The only other method of exploring the upper fornix is more effectual, but also painful. The eye should therefore be well anæsthetized.

(2) The upper lid is everted in the usual manner. A retractor (Fig. 95) is then inserted under the everted lid into the fornix. The margin of the lid being fixed as in (1), the lid is everted a second time, so that the fornix is fully exposed. Sometimes it is necessary to grasp the everted lid with forceps and thus evert it a second time.



FIG. 100. Conjunctival injection in acute conjunctivitis due to an adenovirus.

The redness of the conjunctiva which is observed in irritative and inflammatory conditions varies in its distribution and nature according to the cause. Three groups of blood vessels may be distinguished, though most of them are too small to be recognized in health: (1) the main (posterior) conjunctival vessels running forwards from the fornices (Fig. 100); (2) the anterior extremities of these vessels which form a fine limbal plexus running round the corneo-scleral junction; (3) the anterior ciliary vessels, lying in the subconjunctival or episcleral tissue (Fig. 101). In the last group the perforating branches of the arteries are seen in health as several comparatively large tortuous vessels which suddenly cease about 4 to 5 mm. from the corneal margin. They have numerous small episcleral branches which are invisible in health, but when dilated form a pink zone of fine, straight, closely set vessels around the cornea. The corresponding perforating veins are small, but more numerous than the arteries; their episcleral branches form a closely-meshed network.

Congestion of the individual groups of vessels affords important

evidence as to the seat of disease. The conjunctival vessels can be distinguished from the anterior ciliary by the following points :

(1) They are a brighter brick-red, while the ciliary vessels, being seen through the conjunctiva, have a purple tinge.

(2) If the conjunctiva is moved to and fro over the sclera by placing a finger on the lower lid, the conjunctival vessels also move while the ciliary remain stationary.

(3) The individual vessels and the network they form can be seen in the conjunctival system, whereas the ciliary form for the most part a diffuse reddish-violet blush in which the separate vessels are indistinguishable.

(4) If the blood is pressed out of the vessels by pressure (as by a finger on the lower lid) in a direction away from the limbus, the ciliary vessels fill up at once from the limbus in the wake of the finger on removing the pressure, while the conjunctival vessels fill slowly from the fornix.

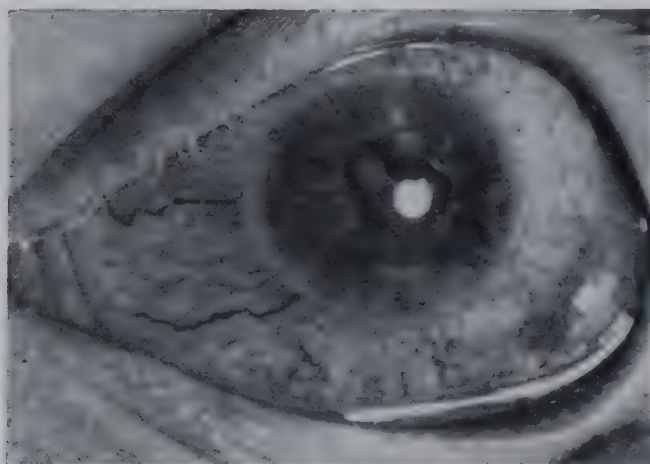


FIG. 101. Ciliary injection in iritis. (Note the deformation of the pupil.)

In general, congestion of the conjunctival vessels, leaving a relatively white zone around the cornea and accompanied by mucous or muco-purulent secretion, is indicative of conjunctivitis. If there is much irritation and photophobia with some blepharospasm and weeping, we suspect the presence either of a foreign body or misplaced lashes or other irritation of the cornea (abrasion, erosions, ulcer, and forms of keratitis). Careful examination shows that the vessels in the circumcorneal zone are bright red, and that the corneal loops of the limbal plexus are also dilated and visible.

In ciliary congestion, on the other hand, which indicates involvement of the inner eye, particularly inflammation of the iris or the sclera, the pink perilimbal injection is supplemented by the dusky lilac tint of congestion of the anterior ciliary vessels (Fig. 101).

These types of congestion, however, are frequently combined so that they then cease to have special diagnostic importance.

Lacrimal Apparatus. Conjunctival congestion of one eye only, or signs of irritation such as watering, should lead us to suspect the efficiency of the lacrimal apparatus. Simple *epiphora* or a flow of tears onto the cheek may be due to malposition of the lower punctum, or to blockage of the canaliculi or nasal duct. Displacement of the lower punctum, which is often very slight, may be easily overlooked but it should be remembered that it is not normally visible without slightly everting the lids. The presence of distension and inflammation of the lacrimal sac should also be noted. This structure is situated in the lacrimal fossa between the inner canthus and the nose, and pressure inwards and backwards at this position will fall upon it. If the sac is distended, the contents—lacrimal fluid, mucus or pus—may regurgitate into the conjunctival sac by way of the canaliculi and will be seen pouring from the puncta. The special methods of testing the patency of the lacrimal passages will be described subsequently (Chap. 32).

The Sclera. Inspection of the sclera around the cornea may reveal the raised congested nodules of episcleritis, while deep scleritis may be shown by dusky ciliary congestion and opacification of the deeper layers of the cornea at the periphery.

Definite blue coloration of the circumcorneal sclera, except in young children, is pathological. It is most frequently due to a herniation of uveal tissue (a ciliary staphyloma) owing to weakness of the sclera (injury, scleritis, etc.) or to increased intra-ocular pressure (glaucoma). Discoloration may be due to pigmentation. Slight duskiness around the points where the anterior ciliary vessels perforate the sclera is not uncommon in people with dark complexions, but otherwise pigmentation in this neighbourhood, either in the conjunctiva or sclera, should be regarded with suspicion as indicative of a melanotic tumour. Definite nodules of deeply pigmented tissue in the situation of the perforating vessels are suggestive of such a tumour of the ciliary body.

The Cornea. A little experience will enable us to recognize at a glance if the cornea is smaller than usual. A small cornea with a shallow anterior chamber is suggestive of glaucoma.

The corneal surface should be bright, lustrous and transparent. Any loss of substance, such as an abrasion, may easily be overlooked without special methods of examination :

(1) Place the patient facing the window. Stand in front and, while directing the patient to follow the index finger which is moved in all directions, observe carefully the image of the window in the cornea (the *corneal reflex*). If the surface of the cornea is normal there will be no distortion of the reflex as it passes over it ; if the surface is roughened the image will be distorted and less clearly defined. This method requires no artificial aid which may not be available in some circumstances.

A more accurate assessment of the corneal surface may be made by *Placido's keratoscopic disc* (Fig. 102), on which are painted alternating black and white circles. The observer looks through a hole in the centre at the corneal image as reflected from a light behind the patient ; a loss in the sharpness of the outline of the image denotes a loss of the normal polish of the corneal surface, while irregularities in the rings betray irregularities on the corneal surface. The image may be photographed to provide an objective record of the optical and anatomical condition of the cornea (Fig. 103).



FIG. 102.
Placido's Disc.

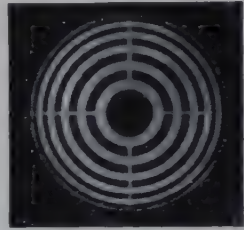


FIG. 103. Photograph
of the normal corneal
reflex ; for an ab-
normal reflex, see
Fig. 181.

(2) To determine the state of the corneal epithelium, the technique of *corneal staining* with a vital dye should be employed by which lesions often minute and invisible to the naked eye are dramatically accentuated in vivid colours. Two dyes are employed (App. I). Fluorescein is the most useful to delineate areas denuded of epithelium (abrasions, multiple erosions, ulcers) which are stained a brilliant green, while Bengal rose stains diseased and devitalized cells a red colour (as in superficial punctate keratitis).

Instillation of the solution of the dye is best done by telling the patient to look downwards ; the conjunctiva above the cornea is then lightly touched with the tip of a glass rod which has been dipped in the solution. A pad of cotton wool is immediately placed upon the closed lids so as to mop up the excess and the tears which tend to flow over the face. If this detail is not attended to the face becomes unnecessarily stained. It is a good plan to wash out any excess of dye with a drop of pantocaine solution, but it is not essential.

Opacities of the cornea may be so faint that they require minute investigation, and the same is true of the details and depth of gross opacities. These can best be studied with the slit-lamp. Of particular importance is the detection of the minute epithelial or subepithelial lesions of a *punctate keratitis* as well as of *keratic precipitates* ("k.p."), small accumulations of cells which adhere to the endothelium and are derived from the uveal tract. The importance of their presence is that they form an indication of inflammation in the uvea, and they are usually associated with an oedematous condition of the corneal endothelium itself (Fig. 104).

In many diseases *new vessels* are formed in the cornea. An exact knowledge of their position, whether superficial or deep, and of their



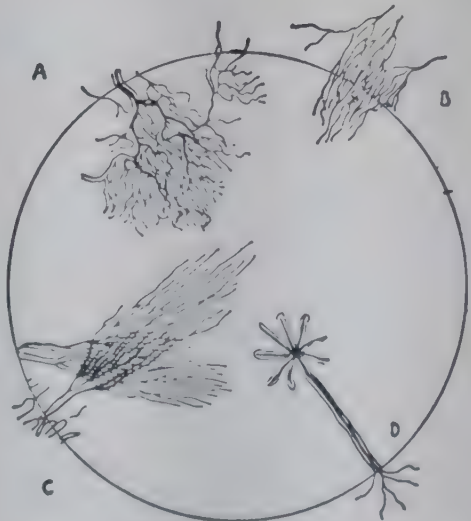
FIG. 104. Keratic precipitates. Seen with the slit-lamp by direct illumination on the posterior corneal surface, and by retro-illumination by light reflected from the iris. The beam of light comes from the right. (From Basil Graves.)

distribution, whether localized, general, peripheral, above, and so on, is of diagnostic importance.

Superficial vessels in the cornea are distinguished from deep (Fig. 105) by the following features :

- (1) Superficial vessels can be traced over the limbus into the conjunctiva, while deep ones seem to come to an abrupt end at the limbus.
- (2) Superficial vessels are bright red and well defined, while deep ones are ill-defined, greyish red, or cause only a diffuse red blush.
- (3) Superficial vessels branch in an arborescent fashion, dichotomously, while deep vessels run more or less parallel to each other in a general radial direction, and branch at acute angles ; their course is determined by the lamellar structure of the substantia propria.
- (4) Superficial vessels may raise the epithelium over them so that the surface of the cornea is uneven, while with deep vessels the cornea, though hazy, is smooth.

FIG. 105. Types of corneal vascularization. A, arborescent (superficial) type ; B, terminal loop type ; C, brush type ; D, umbel type.



The *sensibility* of the cornea may be tested by touching it in various spots with a wisp of cotton-wool twisted to a fine point and comparing the effect with that on the opposite side. Normally there

is a brisk reflex closure of the lids. The sensibility is often diminished in corneal affections, but the change is of diagnostic significance in certain cases, particularly herpes.

The Anterior Chamber. The anterior chamber is shallow in extreme youth and in old age : at other periods of life it is normally about 2.5 mm. deep. It must be remembered that we estimate the depth of the anterior chamber by the position of the iris, and that we view the iris through the cornea, which is a strongly refracting convex surface. The effect of this is to magnify the iris and pupil, and to make them appear farther forwards than they really are. The same applies to anything in the anterior chamber, for example, the point of a knife in operations.

The anterior chamber is usually shallow in closed-angle glaucoma ; abnormally deep in iridocyclitis. It is frequently unequal in depth in different parts. For example, it may be deeper at the periphery than in the centre in iridocyclitis ; on the other hand, when the



FIG. 106. The aqueous flare, showing particles suspended in the anterior chamber as seen by a spot-beam (coming from the right) in a case of cyclitis.

iris is bowed forwards (iris bombé) it is funnel-shaped, the centre being deep, the periphery shallow. Tilting (subluxation) of the lens causes it to be deeper on one side than on the other.

After considering the depth, attention must be paid to the contents. In inflammatory conditions of the uveal tract when the permeability of the vessels is increased, the aqueous may contain particles of protein or floating cells. These are of considerable diagnostic importance. In slighter degrees their presence produces an *aqueous flare* which may be visible only with the slit-lamp when its beam is focused to a point (Fig. 106) ; in its more extreme degrees a turbidity exists easily distinguishable by the loupe. The presence of such an opalescence should, of course, lead to a careful examination of the posterior surface of the cornea to detect whether any protein material or cells are deposited thereon (k.p.). In infected wounds and ulcers of the cornea, and occasionally in iridocyclitis, there is pus in the anterior chamber forming a sediment at the bottom, the surface of which is level (*hypopyon*). A similar collection of blood may occur after contusions or spontaneously (*hyphæma*).

The Iris. The *colour* of the iris and the clarity of its pattern should first be noted. The two irides or parts of the same iris may be of different colour, conditions which are known respectively as heterochromia iridum or iridis. A grey iris with an ill-defined pattern suggests atrophy from cyclitis or glaucoma. Darkly pigmented spots in the iris, not raised above the surface, are common ; care must be taken to distinguish them from small brown or grey raised nodules (melanoma, tubercle, gumma). " Muddiness of the iris " is the expression used for indistinctness of the pattern, caused by inflammatory exudates ; a muddy iris, with small irregular pupil and sluggish reaction to light, is indicative of iritis.

The *position* of the iris must be noted, especially the plane in which it lies. Special attention should be paid to any adhesions (*synechiæ*), anterior (to the cornea) or posterior (to the lens capsule). *Tremulousness* of the iris (iridodonesis) is seen when the eyes are moved rapidly if this tissue is not properly supported by the lens ; this occurs in absence, shrinkage, or dislocation of the lens, slackness of the suspensory ligament, etc. The phenomenon is best seen in a dark room with oblique illumination.

The Lens. The lens cannot be thoroughly examined without the assistance of the slit-lamp and the ophthalmoscope. By inspection, aided by focal illumination, we note any opacities in the pupillary area.

When the light is concentrated by focal illumination upon the pupil of a young person's eye the lens substance seems almost perfectly clear ; at most we see a faint bluish haze. If we examine the lens of an old person in the same manner the haze is much more pronounced ; the lens substance in fact looks slightly milky. We might conclude that the patient has cataract but examination with the ophthalmoscope shows a clear red reflex. The explanation is that the refractive index of the lens substance increases with age, and thus the scattering of light from its surface will be greater ; the milkiness is due to rays of light which are reflected from the lens and enter the eye of the observer.

Opacities in the lens itself are seen by oblique illumination as grey, white or yellowish patches ; with the ophthalmoscope they appear black. According to their distribution and nature we diagnose the various forms of cataract, but our observation must always be confirmed and controlled by ophthalmoscopic examination and the opacities localized by examination with the slit-lamp (Fig. 99). A spot in the centre of the pupil, looking as if it were on the surface of the lens, may be a pupillary exudate or an anterior polar cataract. Triangular spokes of opacity with their apices towards the centre are indicative of senile cataract. A white appearance over the whole pupillary area suggests a total cataract ; if it is yellow and the iris is tremulous we suspect a shrunken calcareous lens. Finally, the pupil may be blocked with uveal exudates (inflammatory pupillary membrane, blocked pupil).

The Pupils. The condition of the pupils should be examined at an early stage in every routine examination of the eyes, certainly before any mydriatic is employed. Such an examination requires careful attention to details and is best carried out as follows :

Place the patient facing the light, which should not be too bright, and arrange so that the two pupils are equally illuminated. Note the size, shape and contour of each pupil. To elicit the *direct reaction to light* cover both eyes with the palms of the hands, preferably without touching the face. While the patient looks straight ahead, remove one hand and watch the pupil, noting if its constriction to light is well maintained. Replace this hand and remove the other, watching the other pupil.

The *consensual reaction to light* is determined by removing one hand so that this eye is exposed to light (it should be shaded from intense light) and watching the pupil as the hand is removed from the other eye. The process is repeated whilst watching the other pupil. The *reaction to convergence and accommodation* is determined by asking the patient to look to the far end of the room. While he does so the index finger is suddenly held up vertically at about six inches from the patient's nose and he is told to look at it. The movement of the pupils is studied while he converges for the finger.

When the reaction to light is feeble and the pupils are already small, it is difficult to be certain of the results in bright diffuse daylight. In such cases the examination should be made in a dark room and light concentrated upon one pupil by focal illumination so that it shines upon the macula, the most sensitive area from which to elicit the light reflex. By slight lateral movements the focus of light can be moved on or off the pupil, the pupillary movements being watched the while. Still finer observations can be made with the slit-lamp, when the microscope is focused on the pupillary margin and the beam is abruptly switched from the side into the pupillary aperture. If there is no movement in these conditions we may conclude that the reaction to light is absent.

The same method will elicit the *hemianopic pupillary reaction* (Wernicke) in the rare cases (lesion of one optic tract) in which it is present. To test for it, the light is placed in front, but rather to one side of the patient. The light is focused on the opposite side of the retina, and the pupil watched. The light is then moved to the other side and is now focused on the other side of the retina. The best source of illumination for this purpose is the focal beam of the slit-lamp reduced to a spot. If the reaction is present the pupil will react briskly when one half of the retina is illuminated, but very slightly when the other half is illuminated. It usually reacts slightly owing to the impossibility of preventing diffusion of light onto the sensitive half of the retina, and for this reason the test is rarely unequivocal.

If these directions are carried out we shall have reliable information as to the shape and relative size of the pupils and their reactions. A few of the commoner conditions may be enumerated here.

Abnormal Size of the Pupil. Dilatation of the pupils with retained mobility is found sometimes in myopia and in conditions of impaired tone or nervous excitement. Conversely the pupils are small in babies and in old people.

Very large pupils will suggest that a mydriatic has been used, perhaps inadvertently as when a patient has been using a liniment containing belladonna, and has rubbed (usually) his right eye with soiled fingers. These pupils are usually immobile, and the patient complains of dimness of vision, especially in near work.

The pupil is also large and immobile in lesions affecting the retina and optic nerve causing blindness (Fig. 35). The most common are complete optic nerve atrophy and absolute glaucoma. In acute glaucoma it is usually large, immobile and oval, with the long axis vertical. It is to be remembered that the presence of a direct reaction to light does not eliminate the possibility of the patient being blind owing to a central lesion affecting the visual pathways above the level of the lateral geniculate body (post-basal meningitis, hæmorrhage, uræmia, etc.).

Dilated and immobile pupils also result from third nerve palsies (*absolute paralysis of the pupil*); if the paralysis also affects the third nerve fibres to the ciliary muscle, accommodation is also paralysed (*ophthalmoplegia interna*). This results from cerebral syphilis affecting the third nerve nucleus, meningitis, encephalitis, diphtheria, lead poisoning, orbital disease or trauma affecting the third nerve or ciliary ganglion or the eye itself.

Unilateral dilatation may result from irritation of the cervical sympathetic as by swollen lymph nodes in the neck, apical pneumonia, phthisis, apical pleurisy, cervical rib, thoracic aneurysm, etc.; it may also be due to syringomyelia, acute anterior poliomyelitis and meningitis affecting the lower cervical and upper dorsal part of the spinal cord and to pressure on the sympathetic fibres leaving the cord in the lower cervical and upper dorsal ventral roots. Many of these causes eventually lead to constriction of the corresponding pupil from sympathetic paralysis. Temporary dilatation of one pupil is not very uncommon.

Small immobile pupils suggest the use of drugs, either locally (miotics), or through the general system (morphia). A small sluggish pupil with muddiness of the iris is associated with active iritis. A small immobile pupil suggests old iritis with posterior synechiæ, and should lead to investigation with a mydriatic such as cyclopentolate to see if the pupil dilates regularly.

Bilaterally small pupils may be due to irritation of the third nerves, arousing suspicion of central nervous disease in their vicinity (as pontine hæmorrhage). The condition can also be due to palsy of the sympathetic, most of the conditions causing an irritative dilatation leading eventually to constriction. When all the sympathetic function on one side is lost, resulting in miosis, a narrowed palpebral fissure and slight enophthalmos (due to loss of tone of Müller's muscle), sometimes with unilateral absence of sweating, the condition is called *Horner's syndrome*.

Abnormal reactions of the pupils are equally important. We have

already seen that loss of the light reflexes results from a lesion in the retina or optic nerve causing blindness and that a hemianopic reaction results from lesions in the tract (Fig. 35). A lesion in the third nerve abolishes both the light and the convergence reflexes.

More complex lesions may result from damage to the relay paths in the tectum between the afferent and efferent tracts. The most important of these is the *Argyll Robertson pupil*, usually caused by a lesion, almost invariably syphilitic, in this region. In it the pupils are small (spinal miosis) and do not react to light, but the contraction to convergence is retained.

The *tonic pupil* (of Adie) somewhat resembles the Argyll Robertson pupil; it is of unknown ætiology, not associated with syphilis, occurs usually in young women, is often unilateral and associated with absent knee-jerks. The tonic pupil is slightly dilated and always larger than its fellow; the unilateral Argyll Robertson pupil is always smaller. Although in the tonic pupil the reactions to light and convergence seem absent at first sight, careful examination shows them to be present although slight and very sluggish with a long latent period. Finally, the tonic pupil dilates well with atropine; the Argyll Robertson pupil does not.

An analysis of the pupillary reactions to drugs frequently allows a differential diagnosis to be made of the causal nerve lesion; the rationale of these tests will be understood from the reaction of the various drugs (p. 39).

If an enlargement of the pupil is due to sympathetic irritation (*spastic mydriasis*), light and accommodation will cause constriction, cocaine will not cause further dilatation since the sympathetic is already stimulated, eserine will cause considerable but not maximal constriction (since the tone of the sphincter overcomes that of the dilatator), and atropine will cause maximal dilatation since the dilatator is now completely unopposed.

If the enlargement is due to paralysis of the third nerve (*paralytic mydriasis*), the reactions to light and accommodation are absent, cocaine and atropine cause a further dilatation and pilocarpine causes constriction if the lesion is proximal to the ciliary ganglion.

If the pupil is small due to irritation of the third nerve (*spastic miosis*), light, accommodation and eserine will cause no appreciable increase in constriction, cocaine a slight and atropine a greater dilatation.

If the miosis is due to sympathetic palsy (*paralytic miosis*), light, accommodation and eserine will cause maximal constriction, cocaine will be inactive and atropine relatively inactive since both muscles are now paralysed.

Gonioscopy. With the methods of examination which we have discussed it is impossible to see into the recesses of the angle of the anterior chamber since this region is covered over by the projecting shelf of the sclera at the limbus. In many conditions such as glaucoma, foreign bodies or tumours, a close inspection of this region is important. It can, however, be made accessible by the slit-lamp provided the beam is diverted at an angle. For this

purpose several types of *gonioscope* have been developed, the simplest of which is that of Goldmann (Fig. 107) whereby a contact lens is inserted between the lids to lie upon the anæsthetized cornea ; the lens is fitted with a mirror in which the image of the recesses of the angle is reflected.

In this way a clear view of the whole of the angle is provided. Fig. 108 shows the main features of the gonioscopic picture. The landmarks from behind forwards are: (1) the anterior surface of the iris; (2) the antero-medial surface of the ciliary body; (3) the trabeculæ covering the canal of Schlemm; (4) Schwalbe's ring (a glistening white line corresponding to the break-up of Descemet's membrane); and beyond this (5), the posterior surface of the cornea which is seen as a convex dome.

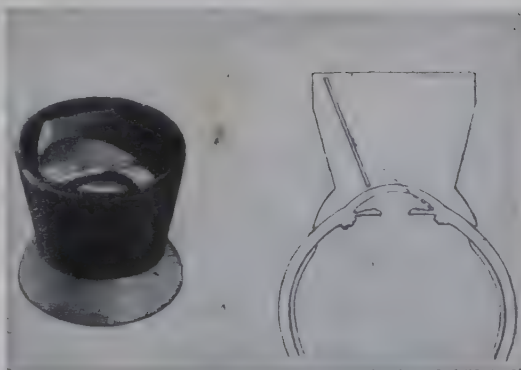


FIG. 107. The Goldmann contact lens used for gonioscopy with the slit-lamp microscope. The figure on the right shows the path of the rays through the lens. They are reflected by the mirror in their path into the angle, and again, as they emerge, into the objectives of the corneal microscope.

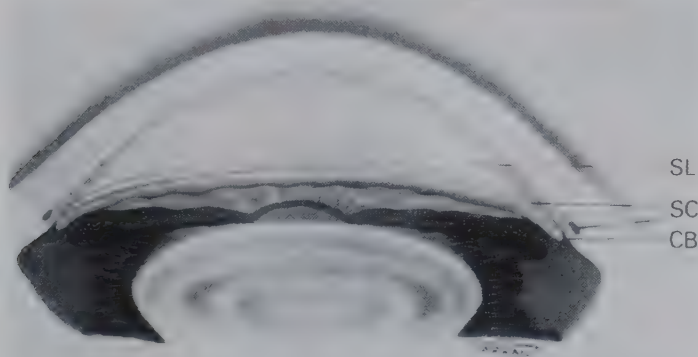


FIG. 108. The angle of the anterior chamber seen gonioscopically.
SL, Schwalbe's line; SC, Schlemm's canal; CB, ciliary band.

Transillumination. In this method of examination an intense beam of light is thrown through the conjunctiva and sclera whereupon the pupil normally appears red ; if, however, a solid mass lies in the path of the light, the beam is obstructed and the pupil remains black. For this purpose, special transilluminators may be employed (Fig. 109) or, more simply, a cap with an open hole at the end may be fitted over the bulb of an electric ophthalmoscope.



FIG. 109 Transilluminator.

In this way a solid mass can be delineated and a tumour differentiated from a cyst. An opaque foreign body can be seen in a cataractous lens, while pupillary reactions may be tested in the presence of a cornea so opaque that accurate vision through it is impossible, although light may be transmitted. Only the anterior half of the eye can be transilluminated in this way, but if there is a mass in the posterior segment of the globe, it can be transilluminated at the time of an operation after the capsule of Tenon has been opened and the transilluminator inserted within it. In the latter type of case a somewhat less reliable method is that of *indirect transillumination*, wherein a powerful source of light is placed in the mouth illuminating the eyes from behind ; normally the pupils have a strikingly luminous appearance, but if a solid mass occupies the fundus, they appear black.

The Tension. Last in the external examination, but by no means of least importance, the tension of the sclera should be assessed ; depending on its degree of rigidity, which varies between individuals, it is increased when the intra-ocular pressure is raised, though not necessarily *pari passu*. It may be done digitally in the same manner as testing for fluctuation in other parts of the body.

The tension may be assessed by the fingers in a somewhat inaccurate manner as follows (Fig. 110). The patient is told to keep looking towards his feet and the index fingers of both hands are placed side by side upon the upper lid above the upper level of the tarsal plate, steadied

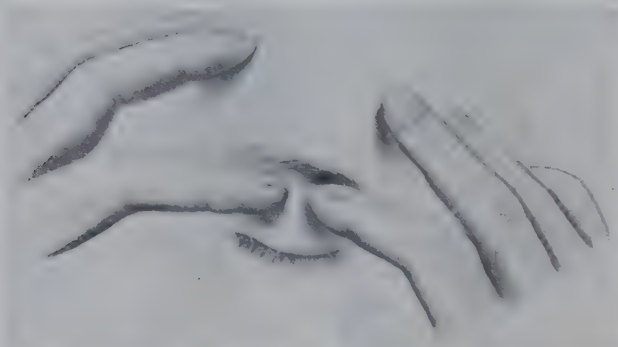


FIG. 110. Digital tonometry.

by the other fingers lightly applied to the brow. One finger is kept quite still, pressing upon the globe through the lid while the globe is indented with the other finger, pressing directly downwards, attention being meanwhile concentrated on the impression of fluctuation which is conveyed to the stationary finger. The normal fluctuation can only be appreciated by practice ; when the eye is very hard fluctuation is absent ; if very soft the response resembles a water-bag.

Instruments known as *tonometers* have been devised for measuring the tension of the intact eye and are of two types. The most common *indentation tonometer* in clinical use is that of Schiötz (Fig. 111); with it the depth of the indentation of the cornea, anæsthetized with 1 per cent. pantocaine solution, made by a weighted stylet is measured by a lever which travels over a scale. There are four weights (5·5, 7·5, 10 and 15 gms.) and the greatest accuracy is attained with the weight which gives a deflection of the lever of 2 to 4 mm. The instrument is calibrated so that the equivalents of the readings in millimetres of mercury can be read off a chart. The readings are inaccurate when transformed into pressures in millimetres of mercury largely because of wide individual variations in the rigidity of the corneo-sclera, but the tonometer is certainly useful to obtain approximate readings and particularly for comparative measurements, as between the two eyes or between successive measurements on the same eye. To allow for this inaccuracy the type of tonometer should always be cited and the reading expressed in this form—20 mm. Hg (Schiötz).



FIG. 111. Schiötz tonometer.



FIG. 112. The applanation tonometer in use. The upper support rests upon the patient's forehead. Below is the applanation tonometer apposed to the eye (Perkins).

An *applanation tonometer* is more accurate than an indentation tonometer since the factor of ocular rigidity is not involved. When the cornea is flattened by the application to it of a plane surface, the intra-ocular pressure is directly proportional to the pressure applied and inversely to the area flattened. The most popular applanation tonometer was designed by Goldmann for use with the Haag-Streit slit-lamp (Fig. 98). In it a flat circular plexiglass plate 7 mm. in diameter is applied to the anæsthetized cornea so as to flatten an area 3·06 mm. in diameter. The constancy of the area is ensured by an ingenious duplicating optical device; a force of 0·1 gm. exerted by a spring-and-lever system corresponds to a pressure of 1 mm. Hg. To avoid the necessity of using a slit-lamp, simpler instruments have been devised based on the same principle (Fig. 112). With these instruments the normal ocular tension is approximately 15 to 18 mm. Hg (applanation).

CHAPTER 11

EXAMINATION OF THE FUNDUS

IN ordinary circumstances the pupil looks black, and no reflex is obtained from the fundus. If, as in Fig. 113, there is a source of light L , in front of the eye, and the eye is focused upon it or accommodated for it, the light and a spot upon the retina are conjugate

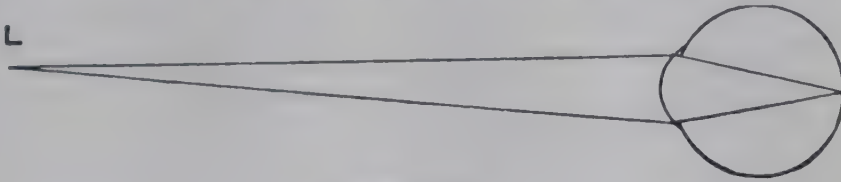


FIG. 113.

foci so that the image of the spot of light is a spot on the retina. Reversing the direction of the rays, all rays from the illuminated spot of the retina are brought to a focus at the source of light. It follows that no rays will enter an observing eye unless it is situated at the source of light. The problem of ophthalmoscopic examination is to make the observing eye at the same time the source of illumination of the observed fundus.

If the eye is not focused for the source of light the conditions are different, and some slight luminosity of the pupil may be seen. This is one cause of luminosity in the pupils of very hypermetropic eyes and in pathological conditions when the retina is displaced forwards as in detachment or by a tumour.

In hypermetropia the conjugate focus of the source of light, L , is a point, l , behind the retina (Fig. 114). Hence the emergent rays from the illuminated

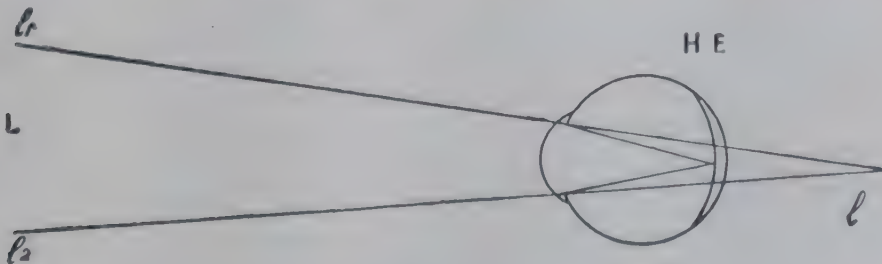


FIG. 114.

area of the fundus are divergent, as if coming from l . Therefore an observing eye situated anywhere within the area $l_1 l_2$ of the cone of emergent rays will catch some of them, and the pupil of the observed eye will appear feebly illuminated. In these circumstances it is not necessary for the observing

eye to occupy the exact position of the source of light, but only a spot in its immediate neighbourhood. On the same principle, the extremely hypermetropic retina pushed forwards by a tumour can be seen well by focal illumination.

The luminosity of albinos' eyes is due to light entering the eye, not only through the pupil, but also through the iris and sclera. That this is the true explanation is shown by the fact that the pupil looks black if it is observed through a small hole in an opaque screen. Only a small amount of light passes through the sclera in the normal eye.

OPHTHALMOSCOPY

It will help us to understand the principles of the ophthalmoscope if we say a few words about its historical development. The ophthalmoscope was invented by Babbage in 1848, but its import-

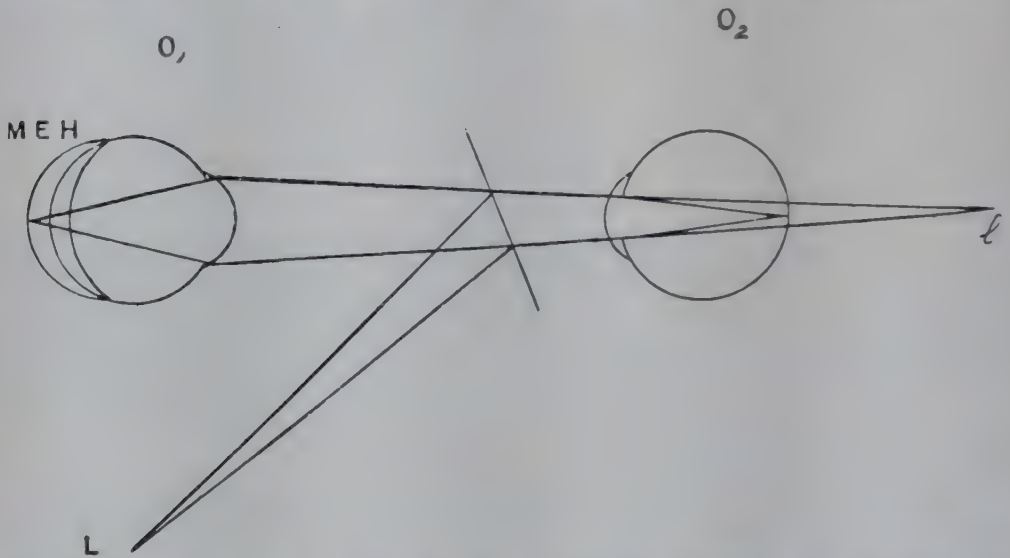


FIG. 115. Diagram of von Helmholtz's ophthalmoscope. O_1 , observed eye; O_2 , observer's eye; L , source of light; l , image of L formed by the plane mirror—immediate source of light; MEH, relative positions of retina in myopia, emmetropia, and hypermetropia respectively, showing the relative sizes of the areas of retina illuminated in each case.

ance was not recognized, and it was re-invented by von Helmholtz in 1850. The original ophthalmoscope of von Helmholtz was merely a plane plate of glass (Fig. 115). A source of light was placed beside the observed eye and the glass plate obliquely in front of it, so that a portion of the light was reflected from the surface of the plate into the eye. On looking through the transparent plate an observer could now receive some of the rays from the fundus into his own eye, and thus obtain an image of the illuminated fundus. Since but a small proportion of the light received upon the plate is reflected at its surface, the illumination is feeble. von Helmholtz next increased the amount of light reflected by superimposing three plane plates. The back of the glass was next converted into a more powerful mirror by silvering it, leaving a small portion unsilvered or a hole in the mirror, through which the observer might look.

The illumination was still feeble, since the rays reflected by a plane mirror are divergent. Reute (1852) therefore introduced the perforated concave mirror which is still generally used. The final modification was the addition of a battery of small lenses of various strengths, which might be brought into position behind the aperture. The many forms of ophthalmoscopes are merely various mechanical contrivances for doing this most conveniently.*

The routine of ophthalmoscopic examination should be as follows:

- (1) Preliminary examination with the plane mirror alone at a distance of about 1 metre from the patient;
- (2) Examination with the mirror alone at a distance of about 22 cm. from the patient;
- (3) Ophthalmoscopic examination by the indirect method;
- (4) Ophthalmoscopic examination by the direct method.

The following facts show the wisdom of this procedure. By (1) we obtain knowledge of the nature of the refraction of the eye under examination; this will prevent many difficulties when we come to closer quarters. By (2) we see any gross changes, especially opacities in the refractive media; these may at once be made evident by this method, whereas they may be very puzzling if first observed by (3) or (4). In addition, we shall see the details of any very hypermetropic part of the fundus, such as a detachment of the retina or a tumour; these also are sometimes by no means difficult to miss by (3) and (4). By (3) we get a general view of the fundus—the largest possible area under moderate magnification; it is comparable to microscopic examination with a low power. By (4) we examine details under a high magnification; it is comparable to microscopic examination with a high power. All these examinations are easier if the pupils are dilated with a mydriatic.

The patient is taken into the darkroom and, if an adjustable self-luminous ophthalmoscope is not used, seated beside a light placed to the side which is to be examined or above his head, but behind the level of his face; the eye should be as much as possible in darkness.

* The student is advised to procure a good ophthalmoscope at the outset of his clinical work in the medical wards. For many years the modification of Couper's ophthalmoscope, generally known as Morton's, was the best instrument of the simple reflecting type; with such a four-mirror ophthalmoscope the surgeon is fully equipped for every detail of ophthalmoscopy and retinoscopy. Various self-luminous ophthalmoscopes have now been devised. In most of these half the aperture of the ophthalmoscope is used for illuminating the fundus from a small electric bulb contained in the instrument, the other half being used for observation. The lamp is run off a small dry battery placed in the handle of the instrument. In the polarizing ophthalmoscope the light is plane-polarized, thus eliminating the troublesome corneal reflex. Self-luminous ophthalmoscopes are particularly useful for examining patients outside the clinic or those who are bedridden, since no separate illumination is required. Some of these instruments, however, have the disadvantage that they are seldom suitable for examination by the indirect method—a method which should never be omitted before using the direct method.

For *examination with the plane mirror alone* the observer sits facing the patient, about a metre from him. He reflects the light from the plane mirror into the eye, meanwhile looking through the sight-hole. An adjustable electric ophthalmoscope or the retinoscope (p. 71) can provide the same illumination. When the light falls on the eye he notices a red reflex from the pupil. There ought to be no black spots in the pupillary area, but either a uniform red reflex or obscure details of the fundus. By tilting the mirror to and fro in various directions he can obtain an approximate idea of the refraction of the eye.

The observer now approaches the patient until his eye, still with the plane mirror, is about 20 cms. from the eye under observation. He can now see the cornea and iris clearly, and can confirm any points which he has made out previously by the external examination.

For *examination by indirect ophthalmoscopy* the examiner then sits again at about a metre from the patient. Keeping the light upon the eye with the concave mirror or the electric ophthalmoscope suitably adjusted, with his left hand he holds a convex lens (about + 13 D) closely in front of the eye, tilting it slightly in order to avoid reflexes. In examining the right eye the patient is told to look at the observer's right little finger which is raised while holding the instrument. In examining the left eye the patient is told to look at the observer's left ear; in this manner the optic disc, which lies a little to the nasal side of the posterior pole of the eye, is brought into the observer's line of vision. He now watches the red reflex from the pupil and, steadying the little finger on the patient's brow, slowly withdraws the lens from the eye towards himself. At a certain point he will see an inverted image of the fundus quite clearly. The indirect method requires practice, but much greater accuracy will be attained if the optical conditions in which the examination is made are thoroughly understood. These will be explained immediately.

Having obtained a good general view of the fundus, the observer again approaches the patient and proceeds to *direct ophthalmoscopy*. Using the self-luminous ophthalmoscope, he looks through the sight-hole and directs the light upon the eye; this is best effected from a short distance away. When the light is on the pupil and the observer can see the red reflex, he slowly approaches until his brow is almost touching the patient's brow. If both the patient and the observer are emmetropic and the latter keeps his accommodation relaxed, he will then probably see the fundus clearly. If either is ametropic, the observer brings into position the appropriate lenses in the ophthalmoscope; and if he cannot relax his accommodation he should use extra concave lenses. The image is erect, the opposite of that by the indirect method. Here again practice is needed, and a knowledge of the optical conditions is essential.

We will now consider the chief features in each stage of the examination.

I. Preliminary Examination with the Mirror at 1 metre.

We will suppose that the observer is emmetropic or that his refraction has been corrected, and that the accommodation of the observed eye is at rest or paralysed by a cycloplegic.

If the eye is emmetropic or has a low refractive error, the rays issuing from any point on the retina are parallel, and since the bundles of rays from two points on the retina diverge after leaving the eye, the observer cannot receive portions of both bundles simultaneously through his pupil (Fig. 116). He

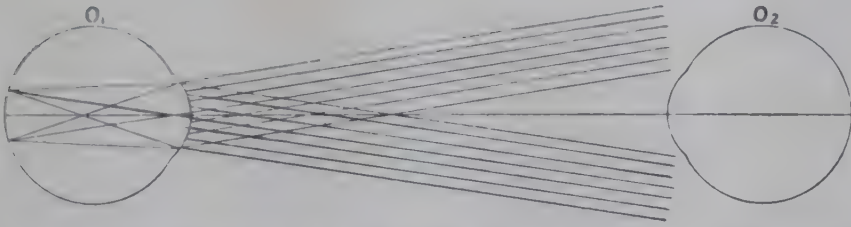


FIG. 116. Examination with the mirror at 1 metre. O_1 , observed eye, which is emmetropic ; O_2 , observer's eye : none of the rays from the widely distant points on the fundus of O_1 enters O_2 . If the points are close together the rays of the two bundles will be nearly parallel, and would form a clear image on the retina of O_2 if the accommodation of O_2 were almost completely in abeyance.

cannot therefore see two spots on the retina but can only focus one at a time, and thus sees only a general illumination. In the hypermetropic eye, however, the emerging rays are divergent, and the two bundles of rays from two points on the retina will form two divergent bundles (Fig. 117). These will appear to come from two points behind the eye where an imaginary erect image is formed. Since each bundle diverges, some of the peripheral rays of each will be received by the observer's pupil, so that he will obtain a clear image of each point and thus see the virtual image behind the patient's eye.

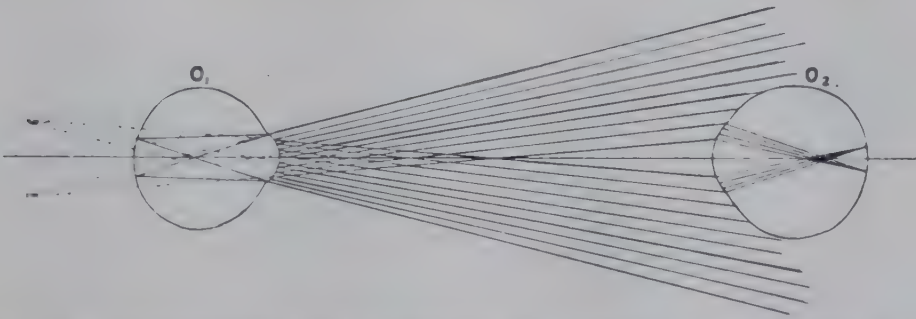


FIG. 117. Examination with the mirror at 1 metre. O_1 , observed eye, which is hypermetropic ; O_2 , observer's eye, emmetropic, but accommodated for the divergent rays from O_1 .

In myopia, on the other hand, the rays coming from the two points will be convergent and will form a real inverted image in front of the eye (Fig. 118). Continuing from this image the rays will diverge in two bundles, the peripheral parts of which will enter the observer's pupil. He will thus see a small inverted image of the fundus. If the observer now moves his head from side to side, the erect image in hypermetropia will appear to move in the same direction, and the inverted image in myopia in the opposite.

It therefore follows that in the preliminary examination with the plane mirror, if the fundus reflex is seen as a uniform red glow (the

red reflex), the eye is emmetropic or approximately so ; but if any details of the retinal structure are seen, a considerable degree of ametropia exists. If the picture thus presented appears to move in

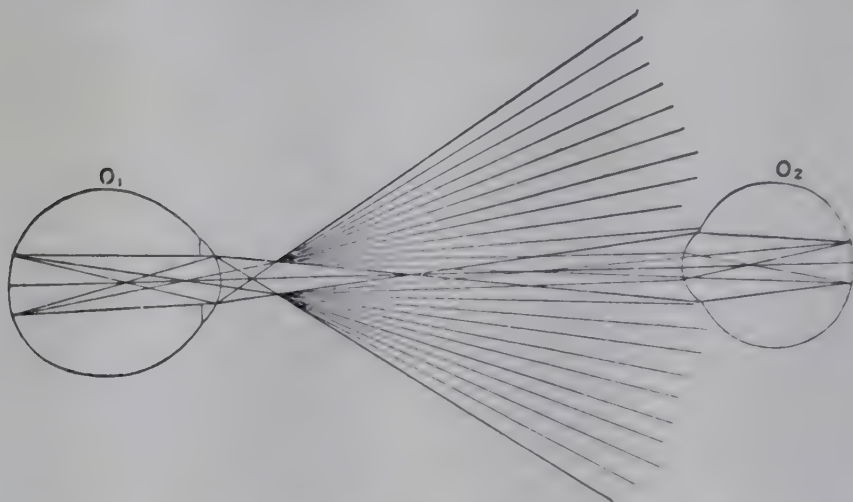


FIG. 118. Examination with the mirror at 1 metre. O_1 , observed eye, which is highly myopic ; O_2 , observer's eye, emmetropic, but accommodated for the divergent rays from the far point of O_1 .

the same direction as the observer's head, the refraction is hypermetropic ; if it moves in the opposite direction, it is myopic. This may be verified and a more accurate assessment of the refraction gained by retinoscopy.

II. Preliminary Examination with the Mirror at the convenient distance for near vision (22 cm.). At this distance the observer will be most suitably situated for distinct unaided vision, and he will be able to examine the superficial parts of the eye more accurately. If he is hypermetropic or presbyopic he will naturally have to use a convex lens. The purposes of a preliminary examination in this manner are (1) the recognition of opacities in the refractive media ; (2) the recognition of a detached retina or other structure not far behind the lens ; (3) the confirmation of the results found by the external examination.

(1) *The diagnosis of opacities in the refractive media.* If there is any opaque body in the course of the rays reflected from the fundus it will stop these rays and will therefore appear black. The whole field may be black, as when the lens is entirely opaque, or when there is blood in the vitreous. If small opacities are seen their motility is determined by telling the patient to turn his eye in different directions ; a floating opacity will then continue to move after the eye is brought to rest, in which case it must be either in the aqueous or vitreous which latter in this event must be fluid. The exact position may be determined by observing its *parallactic displacement*.

In Fig. 119, if 4 is the centre of rotation of the eye, and if there are opacities at 1, 2, 3, 4, 5, then, when the eye is rotated a small amount, all the opacities except 4 will move, the amount of movement being greater the farther the opacity is from the centre of rotation. Since all the movements will be referred to the edge of the pupil for comparison, to an observer situated at A, all the

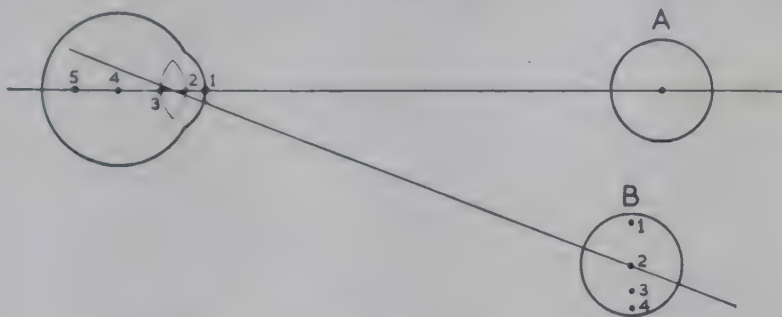


FIG. 119. Parallax displacement.

opacities will appear as a single spot in the centre of the pupillary reflex. If he shifts his position to B, or if the eye is rotated in the opposite direction, the opacity 2 will remain in the centre of the pupil, whilst 1 will appear to move towards one edge of the pupil, and 3, 4 and 5 towards the opposite edge, 5 being lost behind the iris.

Hence we deduce the rule that if the eye is moved slightly in a given direction, opacities in the pupillary plane will appear stationary ; those in front of that plane will move in the same direction, and those behind will appear to move in the opposite direction, the amplitude of apparent movement being a rough indication of their distance from the pupillary plane.

The *corneal reflex*, the image of the mirror formed by the cornea, can also be used as a guide. It is a virtual image situated about 4 mm. behind the anterior corneal surface, that is, a short distance behind the anterior surface of the lens (behind 2 in Fig. 119). The centre of curvature of the cornea is situated 8 mm. behind its anterior surface (less than 1 mm. behind 3 in Fig. 119). By this method of examination the corneal reflex will always cover this latter spot, the centre of curvature of the cornea, no matter what the position of the eye. Hence an opacity situated here will always be covered by the corneal reflex ; opacities in front of the corneal reflex move in the same sense with regard to the reflex as the eye moves ; and opacities behind it move in the opposite direction to the movement of the eye.

This method of examination affords the surest means of discovering the edge of a *dislocated lens*, or the notch in the edge of the lens in congenital *coloboma of the lens*. When the edge of the lens crosses the pupillary area it is seen as an intensely black crescent, since the whole of the light reflected from the fundus which falls upon the extreme edge of the lens is totally reflected within the lens ; none of it leaves the eye, so that none of it can enter the observer's eye.

(2) *The recognition of a detached retina or a tumour arising from the fundus.* We have already discussed the optical conditions rendering such lesions visible, the retina being in the position of the fundus of a very hypermetropic eye. When light is thrown in from the mirror a difference of reflex in different directions is noticed, red in some, grey

or black in others. More minute investigation will reveal a whitish or greyish uneven surface upon which the retinal vessels are seen as black wavy lines, an important observation since the appearance of a detached retina by ophthalmoscopy may be puzzling to the beginner.

(3) *Confirmation of the results found by the external examination.* We are able by this method not only to confirm the results previously arrived at by external examination, but also to supplement some of them by important subsidiary information. Thus we are able to map out the limits of opacities in the lens much more accurately, since they now appear black on a red background. A black spot in the iris may allow a red reflex through it and thus show itself to be a hole. Similarly a black patch at the ciliary margin of the iris may be a melanotic tumour of the ciliary body growing forwards and implicating the iris : or it may be a separation of the iris from its ciliary attachment (iridodialysis) ; in the latter case it will be possible to obtain a reflex through it by the mirror, whereas in the former it will be opaque.

By this method, also, superficial opacities, such as those in the cornea and near the anterior surface of the lens, can be seen in their natural colours at high magnification by approaching still nearer to the eye and using stronger convex lenses behind the mirror. Thus if we approach very closely to the eye and place a $+20$ D lens behind the mirror the opacities in the cornea are seen highly magnified, while those in the lens may be brought more clearly into focus with a slightly weaker lens.

III. The Indirect Method. The indirect method of examination with the ophthalmoscope consists essentially in making the eye, whatever be its refraction, highly myopic by placing a strong convex lens in front of it so that a real inverted image of the fundus is formed between the observer and the convex lens (Figs. 120-1). In all cases the image is magnified, the amount of magnification depending upon the refraction of the eye, the strength of the lens

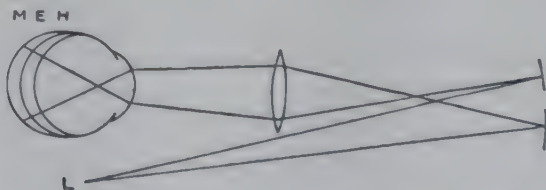


FIG. 120. Indirect method. Illumination of the fundus, showing the course of rays from the source of light to the mirror, through the lens, and through the eye ; also the area of the field of illumination.

and its distance from the eye. With a lens of $+13$ D the fundus of an emmetropic eye is magnified about five times.

It will be seen that with the same lens the inverted image is formed at different distances beyond it according to the refraction of the eye. If the lens is kept at a constant distance from the eye, for example, its own focal distance, the emmetropic image will be formed at the focal distance of the lens beyond it : the myopic will be nearer to the lens, the hypermetropic farther from it (Fig.

122). Since in all cases the image is formed in the air between the lens and the observer's eye, the observer must not approach too closely to the patient (a natural impulse in order to see the aerial image clearly with his unaided eye); if he does so he will find it necessary to put up an appropriate convex lens, which, incidentally, will magnify the image.

One of the difficulties in the indirect method is the reflexes formed by the eye and the surfaces of the lens. The cornea forms a reflex of the mirror which, when seen through the convex lens, is magnified, so that it may cover the pupil and prevent anything behind being seen. The surface of the lens towards the observer acts as another convex mirror and forms another reflex situated behind the lens. Similarly the surface of the lens near the patient acts as a concave mirror and forms a reflex on the observer's side of the lens. These reflexes are troublesome, but they may be got out of the way by tilting the lens so that they move in opposite directions and a view is obtained between them. The tilting should not be overdone since the optical effect of astigmatism is produced and the fundus appears distorted.

Theoretically, to obtain the maximum field, the best place for the lens is its own focal distance from the patient's pupil; but this is the worst place for the corneal reflex. Since the latter is situated near the level of the iris, if the convex lens is at its focal distance from it, the rays from this image will be made parallel by the lens and the reflex will fill the whole area of the lens so that nothing else is seen. The best position for practical purposes is either nearer to or farther from the eye than this, and the convenient distance is where the lens is at its focal distance from the anterior focus of the eye. Here, slight tilting of the lens, besides shifting the lens reflexes out of the way, will also move the corneal reflex and the image of the fundus in opposite directions, allowing an uninterrupted view.

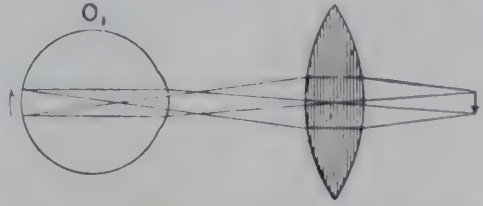


FIG. 121. Indirect method. Emergent rays from the fundus, showing the formation of the image. In the figure the lens is situated at the anterior focal plane of the eye; the rays which are parallel inside the eye, therefore, pass through the optical centre of the lens. The rays which pass through the nodal point of the eye are rendered convergent by the lens. The points where these two systems of rays cross give the position of the image, which is seen to be inverted.

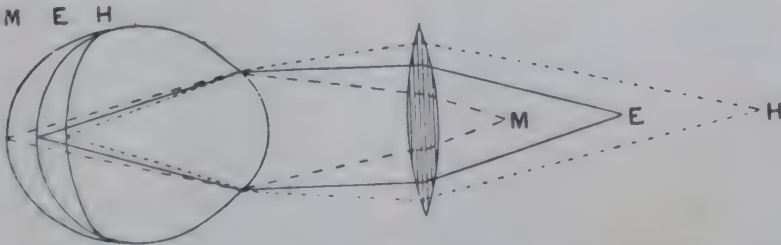


FIG. 122. Indirect method. Position of the image according to the refraction of the eye. In this figure the lens is situated at its own focal distance from the cornea. In emmetropia the parallel emergent rays, therefore, cross at the principal focus of the lens at E. In myopia the convergent emergent rays cross nearer to the lens than its principal focus, at M; in hypermetropia the divergent emergent rays cross farther from the lens than its principal focus, at H.

Differences of level of two points near each other on the fundus are made very evident by parallaxic displacement with the indirect method. Thus, in Fig. 123, if there are two points, *a* and *b*, at different levels in

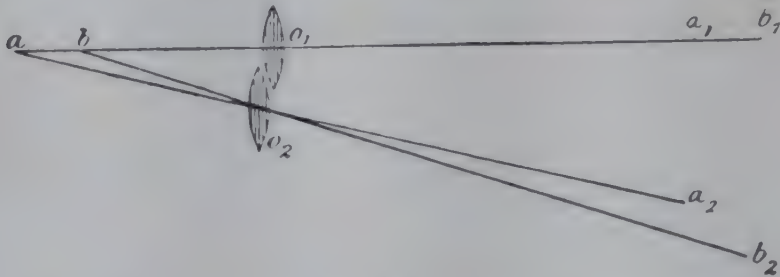


FIG. 123. Indirect method. Parallaxic displacement.

the fundus, for example, on the edge of the disc and at the bottom of a glaucomatous cup, when the lens is shifted slightly so that its optical centre moves from o_1 to o_2 , the images of *a* and *b* will move from a_1 to a_2 and b_1 to b_2 .

IV. The Direct Method. In the direct method the patient's eye is approached as closely as possible by the observer whose eye thus receives the emergent rays from the fundus directly (Fig. 124).

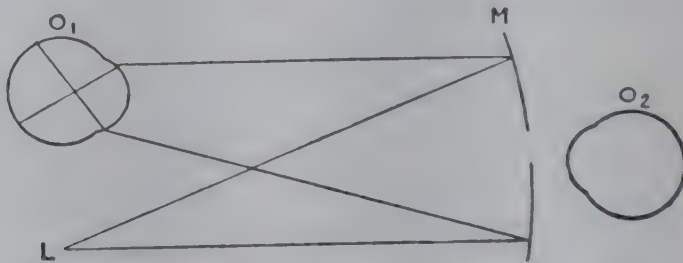


FIG. 124. Direct method. Illumination of the fundus, showing the course of rays from the source of light to the mirror and through the eye; also the area of the field of illumination. Compare with Fig. 120.

If the patient is emmetropic (E, Fig. 125), the issuing rays will be parallel, and will be brought to a focus on the retina of the observer. If he is hypermetropic, the emergent rays will diverge (H, Fig. 125), and consequently will only be brought to a focus on the observer's retina if he accommodates, or by the help of a convex lens. If he is myopic, they are convergent (M, Fig. 125) and must be made more divergent by the interposition of a concave lens if a similar focus is to be formed. In emmetropia, therefore, the image of the retina is seen clearly without any lens in the ophthalmoscope; in ametropia, in order that the image be clearly seen, a lens corresponding to the refractive error must be used. If, however, the eye is very highly myopic its punctum remotum will be situated somewhere in space between the eye itself and the observer's ophthalmoscope so that

it may be impossible to obtain a clear image with any correction ; in such cases a view of the fundus may be possible if the patient's spectacles are left in place and the examination is made through them.

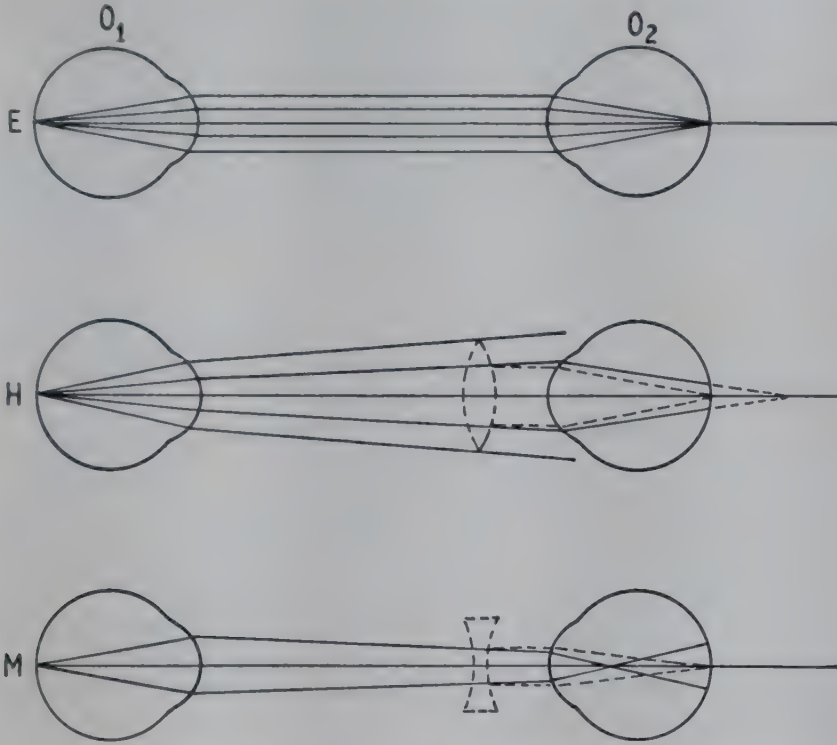


FIG. 125. Direct method. Emergent rays from the fundus of the observed eye, O_1 , showing the formation of the retinal image on the retina of the observer's eye, O_2 . In emmetropia, E, the emergent parallel rays are brought to a focus on the retina of O_2 if the accommodation of this eye is absolutely at rest. In hypermetropia, H, the emergent divergent rays are brought to a focus on the retina of O_2 , either by means of accommodation or by placing a convex lens in front of O_2 . In myopia, M, the emergent convergent rays can only be brought to a focus on the retina of O_2 by placing a concave lens in front of O_2 .

Much stress is generally laid upon the necessity and the difficulty of relaxing one's accommodation in examination by the direct method. It is difficult to relax the accommodation entirely when the eye is apparently close to the object looked at. The observer should try to think that he is looking at a very distant object, but even then, as soon as he directs his attention to details of the picture, he is almost certain to accommodate. It is best for the beginner not to worry himself about this point : if he cannot see an emmetropic fundus clearly, let him put up minus lenses until he does. When he has acquired facility in seeing anything at all it will be soon enough for him to grapple with this difficulty.

The image by the direct method is always erect and is also more magnified than by the indirect method. In emmetropia the fundus is seen magnified about fifteen times, somewhat less in hypermetropia and more in myopia.

The area of the fundus which can be seen by the direct method varies with the distance of the observer from the eye and with the refraction. It increases as the eye is approached, is greatest in hypermetropia, least in myopia, and intermediate in emmetropia. Thus, we see the largest area, least magnified, in hypermetropia, and the least area, most magnified, in myopia. In astigmatism the magnification is greatest in the more myopic meridian, and least in the more hypermetropic so that there can be no clear image of the whole field. *Only lines perpendicular to the meridian which is corrected are seen clearly.*

If there is a difference in level between two points on the fundus, it is made manifest in the direct method also by parallax displacement if the observer moves slightly to one side ; an object farther forward always moves in the opposite direction to the movement of the observer's head.

The difference in level can be accurately measured. Thus the bottom of a cupped disc will be relatively myopic to the edge so that a more concave lens will be required to see the vessels at the bottom of the cup clearly, while the top of an eminence, such as a swollen disc or a tumour, will require more convex lenses than are needed to see clearly a blood vessel on a normal part of the retina near the disc. It can be proved that *if the correcting lens is at the anterior focus of the emmetropic eye* a difference of 3 D is equivalent to approximately 1 mm. difference of level at the fundus. It is important to get as close as possible to the eye when measuring differences of level and to relax the accommodation, because only then are the conditions of accuracy fulfilled. In order to eliminate the effect of the observer's accommodation the lowest minus lens or the highest convex lens which allows clear vision must be chosen for the purposes of measurement.

An opacity in the vitreous provides the same optical conditions as the fundus of a hypermetropic eye. Such an opacity can be examined by putting up convex lenses until it is clearly focused ; by using convex lenses from 0 to + 20 D and by withdrawing slightly from the eye, we can thoroughly explore the emmetropic eye from the fundus to the surface of the cornea. The appearance of opacities in the vitreous or lens will vary with their density and with the amount of light reflected from their surfaces ; if they are very dense they will appear black against the background of the red reflex, but if they are semi-transparent they will appear red or whitish according to the relative amounts of light transmitted from the fundus and reflected from their surface. A detached retina may therefore look red or white according to its degree of transparency, and if much light is reflected from the surface details may be seen upon it.

EXAMINATION OF THE FUNDUS BY FOCAL ILLUMINATION

The ordinary slit-lamp, as we have seen, cannot be used to explore the eye further back than the anterior parts of the vitreous because the beam of light is ordinarily brought to a focus in this region. If, however, the beam is made more divergent by eliminating the refrac-

tive influence of the corneal curvature by using a contact lens with a flat anterior face, or (more simply) by interposing a — 55 D lens in front of the cornea, the posterior part of the vitreous and the central area of the fundus can be examined by the binocular microscope in the focused beam of light. Such an examination requires full mydriasis. By this method fine changes in the posterior part of the vitreous and in the retina and at the optic disc can be readily studied, areas of œdema are clearly outlined in the optical section, and difficult problems in diagnosis such as the difference between a cyst or hole at the macula are clearly demonstrated (Plate IV, Figs. 3 and 4).

CHAPTER 12

THE FUNDUS OCULI

WHEN the fundus is seen it appears a bright red colour : this is due chiefly to the blood circulating in the choroid. In people of dark complexion no choroidal blood vessels are evident on account of the retinal pigmentary epithelium which, while dense enough to blur any details, is not sufficiently so to prevent the colour of the blood manifesting itself.

The Optic Disc. The first object to be sought is the optic disc (Plate III, Fig. 1). It is pale pink in colour, the tint showing considerable variations within normal limits. It is nearly circular but seldom perfectly so ; it is about 1.5 mm. in diameter, but, of course, is seen magnified. An oval appearance due to astigmatism must be borne in mind. The edges are usually sharp, but sometimes a little irregular and certain physiological variations without pathological significance should be noted. Not uncommonly, especially in old people, there is a narrow white ring around the pink disc, the *scleral ring* ; this is due to the fact that the choroid and the pigmentary epithelium of the retina do not extend quite up to the margin of the disc so that the sclera is seen through the retina. Sometimes there is a ring of black pigment around the margin of the disc due to the heaping up of the retinal pigmentary epithelium. More commonly parts of the circumference have black patches, but they are not continuous.

The disc itself is seldom uniformly pink. The central part is usually paler and may be quite white, and this lighter area may extend nearly but rarely quite to the temporal edge of the disc so that the temporal side is normally paler than the nasal. The central vessels emerge from the middle of this white area, usually from a funnel-shaped depression, the *physiological cup*. This cup varies in different eyes. When it is deep the central part may be seen to be speckled with grey spots representing the meshes of the lamina cribrosa through which the nerve fibres pass. Sometimes there is scarcely any physiological cup, in which case the disc is more uniformly pink and the central vessels may have already divided before they come to the surface. The true nature of the physiological cup is best understood by comparing the ophthalmoscopic picture with a microscopic section vertically through the nerve-head (Fig. 126).

The colour of the disc is due to the white fibres of the lamina cribrosa seen through the vascularized nerve tissue. Where the nerve fibres are thinnest, in the centre, the white lamina shines more brightly through. The grey spots in the lamina, when they are seen, are due to the non-medullated nerve fibres reflecting less light than the white connective tissue fibres.

The Retinal Vessels. The retinal vessels are derived from the central artery and vein, which usually divide into two branches at or near the surface of the disc to form a superior and an inferior trunk (Plate III). Each trunk usually divides into two, one of which sweeps up (or down) towards the temporal side, the other up (or down) towards the nasal side—the superior and inferior temporal and nasal arteries and veins. These divide dichotomously into innumerable branches the mode of division being subject to great variations, but the nasal branches run more radially than the temporal, which make a decided sweep to avoid the macula.

The arteries are distinguished from the veins by being lighter red and narrower. The veins have a purplish tint and are often more convoluted although less frequently the arteries are tortuous. Ophthalmoscopically the blood column is seen, not the vessel wall which is normally transparent. Each, but especially the arteries, may have a bright silvery streak running longitudinally down the centre, due to reflection of light from the convex cylindrical surface.

The Macula lutea (Plate IV, Fig. 1) is situated about 3 mm. or 2 disc-diameters (2 d.d.) to the temporal side of the edge of the disc, a little below the level of the horizontal meridian. It is difficult to see without a mydriatic unless dim illumination is used, for the bright light on this most sensitive spot causes maximal constriction of the pupil: the corneal reflex then often obliterates all view. It varies in appearance according to illumination, refraction and complexion. In general, it is a small circular area of a deeper red than the surrounding fundus, and in its centre there is nearly always a *foveal reflex*, due to reflection of light from the walls of the foveal depression. This is most frequently seen as a silvery ring of light hiding everything behind it: it may be circular or oval, according to the incidence of the light and the refraction of the eye. Often there is an intensely bright spot at or close to the fovea, also due to reflection.

The macular region is supplied by twigs from the superior and inferior temporal arteries, and by small branches coming straight from the disc. There are no retinal blood vessels at the fovea itself, and none can be seen ophthalmoscopically for a little distance around. Occasionally small arteries (cilio-retinal) derived from the



FIG. 126. Physiological cup.
R, retina; P, pigmentary
epithelium; C, choroid;
S, sclera.

ciliary system start near the edge of the disc, run inwards, and then bend sharply outwards towards the macula.

The General Fundus. The appearance of the general fundus varies considerably in health. In people who are neither very dark nor very light in complexion the spaces between the retinal vessels show a uniform redness, occasionally with a very delicate punctate stippling, especially towards the periphery. In albinos the choroidal vessels are seen clearly, the spaces between them being white where the sclera shines through. In partial albinos in whom the hair is very fair in infancy, the macular region usually shows a uniform normal redness, the lack of pigmentation being manifested peripherally. In very dark people the fundus is a darker red, and indications of the choroidal vessels are often seen as indefinite brighter red streaks. Sometimes the pigment between the choroidal vessels is particularly dense, or the pigment is deficient in the retinal pigmentary epithelium, while the choroid is deeply pigmented: the choroidal vessels are then seen to be separated by deeply pigmented polygonal areas (*tigroid* or *tesselated fundus*) (Plate III, Figs. 2 and 3).

There is no difficulty in distinguishing the choroidal from the retinal vessels when both are visible (Plate III, Fig. 3). The former are broader and ribbon-like, without any central reflex streak: they anastomose freely, whereas the retinal vessels do not anastomose at all. Moreover, in certain parts, their anatomical distribution is characteristic (p. 11).

The order of examination of the details of the fundus should be systematic. Applying the indirect method we obtain a general view. The patient is instructed to fix his gaze in such a direction that the disc is brought into view (p. 116). The shape and colour of the disc, the arrangement of the vessels, the colour of the choroidal reflex (its uniformity or tessellation), gross abnormalities (white or pigmented spots, etc.), are readily noted. The patient is then directed to look up to the ceiling, to the right, to the left, and down to the ground; in the latter position the upper lid is gently raised by a finger of the hand which is holding the large lens, as otherwise it will cover the pupillary area. In this manner the periphery of the fundus is brought into view. Even when the central parts of the fundus are uniformly tinted the periphery often displays traces of the choroidal vessels, associated with greater pigmentary stippling or a diminution of pigment. Only minute investigation with the direct method can show whether this is normal or pathological.

It cannot be too strongly emphasized that examination by the indirect method should not be neglected. The topographical distribution of lesions in the fundus is often more important for diagnostic purposes than minute details. The introduction of self-luminous ophthalmoscopes has unfortunately tended to discourage indirect ophthalmoscopy.

PLATE III
VARIATIONS OF THE FUNDUS



FIG. 1. Normal fundus.



FIG. 2. Tigroid fundus.

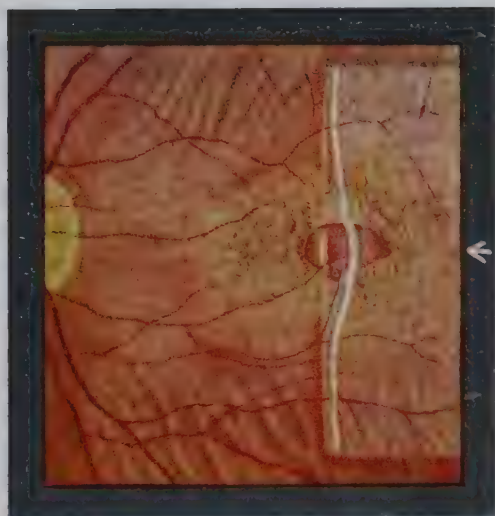
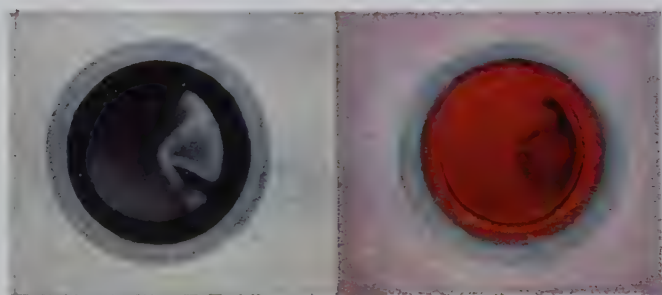


FIG. 3. Albinotic fundus.



FIG. 1. The normal macula.

FIG. 2. A zonular cataract seen by reflected (focal illumination) and transmitted light (ophthalmoscopically).



FIGS. 3 and 4 Slit-lamp examination of the fundus. Fig. 3, retinal cyst at the macula : Fig. 4, hole at the macula. (H. Goldmann, *Brit. J. Ophthalm.*)

Having thus obtained a good general idea, the systematic examination is repeated by the direct method, paying special attention to points which the indirect method has left uncertain—the details of the disc and the vessels as well as hæmorrhages, white spots of exudate and other pathological appearances.

The macula is next examined. It may be brought into view by telling the patient to look into the light ; but it is best to fix the temporal edge of the disc and pass horizontally outwards for a distance of about two disc diameters (a convenient unit in ophthalmoscopic topography), when the macula will be found. If the patient is not under a mydriatic or the pupil is not large the light should be lowered so that the constriction of the pupil may be reduced to a minimum. The corneal reflex is always troublesome, but has to be dodged. Any abnormality at or near the macula is of the utmost importance. Black or white spots are often difficult to distinguish from shadows or reflexes: if either has a sharp contour, and if they do not seem to shift when a minute movement is made with the ophthalmoscope, it may be concluded that they are pathological entities.

Finally, the periphery of the fundus is investigated. With full dilatation of the pupil it is possible to see almost to the ora serrata, particularly with the indirect method of ophthalmoscopy and especially if the sclera over the ciliary region is slightly indented with a glass rod after the eye has been lightly anæsthetized. The periphery, even in an emmetropic eye, is usually best seen with a low convex lens, owing to the obliquity of the axis of the rays as they pass through the crystalline lens.

A binocular ophthalmoscope supported on the forehead and carrying its own source of (powerful) illumination for indirect ophthalmoscopy is of great value in the analysis of fine details of the retina, particularly in the periphery. It is especially useful in the search for small retinal holes in cases of detachment.

Vascular Pulsation. In normal conditions no pulsation can be seen in the retinal arteries. In some 10 to 20 per cent. of people, however, retinal *venous pulsation* may be seen at or near the edge of the disc or, indeed, wherever veins take a very sharp bend ; we have already noted that this is due to transmission of the intra-ocular pressure. The venous pressure is lowest at the point near the disc, and there is a certain amount of obstruction to the flow of blood as the vessels pass through the narrow neck at the lamina cribrosa. With each arterial pulsation the intra-ocular pressure is suddenly slightly raised so that the increased pressure on the outside of the walls of the veins tends to make them collapse. This causes a sudden increased obstruction to the outflow of blood from the eye during systole, but the venous circulation recovers itself during the arterial diastole. The venous pulsation can be increased or made manifest if absent by increasing the intra-ocular pressure by slight pressure with the finger on the globe. It will be noticed that it is diastolic and therefore has been called the *negative venous pulse*.

Two other forms of venous pulse occur in pathological conditions.

The *positive venous pulse* is pre-systolic, continuing into the systolic phase : it is due to tricuspid regurgitation, and is permitted by the normal insufficiency or absence of valves in the jugular veins. The *transmitted centripetal venous pulse* is an accentuation of the normal tendency of the pulse wave to progress through the capillaries into the veins, owing to the intra-ocular pressure. It is due to venous congestion, with or without increased *vis a tergo*.

Visible *arterial pulsation* is always pathological. Since the pressure in the central retinal artery is far above the intra-ocular pressure, it would not be surprising if the pulse wave were transmitted and could be seen. Normally, however, the intra-ocular pressure damps the pulsation, and the increase in pressure which accompanies each pulsation is spread over the whole volume of the contents of the globe and is transmitted to the plastic sclera ; such pulsations as survive this damping effect are too slight to be observed in such small vessels by ordinary ophthalmoscopic examination.

Two types of arterial pulsation occur pathologically : (1) a true pulse wave, accompanied by locomotion of the vessels ; (2) an intermittent flow of blood or pressure pulse. The true arterial pulse occurs in such conditions as aortic regurgitation, aneurysm or exophthalmic goitre ; it is not confined to the disc. In the pressure pulse the arteries fill only with the heart-beats, being empty between them ; it is only visible on the disc, and may be produced in a normal eye by external pressure upon the globe by a finger applied to the lid. This type of pulsation is purely a pressure phenomenon, and is caused by a considerable increase of intra-ocular pressure with normal or lowered blood pressure, as in glaucoma, or by any considerable diminution of blood pressure with normal intra-ocular pressure, as in syncope or orbital tumours.

Capillary pulsation is seen only in aortic regurgitation as a systolic reddening and diastolic paling of the disc.

Ophthalmodynamometry. In view of the fact that the central retinal artery is a branch of the ophthalmic artery, itself a branch of the internal carotid just at its termination, the arterial pulse seen at the optic disc when the intra-ocular pressure is raised can be used to assess the pressure in this important vessel particularly on a comparative basis between the two sides. If the intra-ocular pressure is raised sufficiently to stop the circulation in the artery at the disc, we are measuring the lateral pressure of the ophthalmic artery which is virtually the pressure of the internal carotid. A spring-loaded plunger calibrated in G. of pressure exerted (*dynamometer*) is pressed against the sclera of the anæsthetized eye over the insertion of the lateral rectus, and the disc is observed with the ophthalmoscope. The pressure is increased until the circulation in the central artery is obliterated ; as the pressure is lessened the first sign of pulsation to reappear gives the systolic pressure in the ophthalmic artery ; and as the pressure is further lessened the maximal pulse gives the diastolic pressure. These readings can be converted into mm. Hg of intra-ocular pressure with conversion tables. A unilateral fall of pressure of more than 20 per cent. suggests a stenosis or occlusion of the carotid system proximal to the origin of the ophthalmic artery ; in cases of aneurysm a low diastolic ophthalmic pressure indicates that carotid ligation might be hazardous ; while in glaucoma an approach of the intra-ocular pressure to the diastolic pressure indicates that the margin of safety before retinal damage may occur is small.

CHAPTER 13

EXAMINATION OF RETINAL FUNCTION

THE functional examination of the eye consists of testing the acuity of the forms of visual perception which have already been mentioned—the light sense, the colour sense, and the form sense. They are usually tested in the reverse order. Each eye must be tested separately throughout.

The Acuity of Vision. The acuity of *distant central vision* is now almost invariably tested by means of Snellen's Test Types (Fig. 127). These are constructed upon the standard that the average minimum visual angle is 1 minute.

The types consist of a series of letters arranged in lines each diminishing in size. The breadth of the lines of which the letters are composed is such that the edges will subtend an angle of 1 minute at the nodal point of the eye at a particular distance. Each letter is of such a shape that it can be placed in a square the sides of which are five times the breadth of the constituent lines. Hence the whole letter will subtend an angle of 5 minutes at the nodal point of the eye at the given distance (Fig. 128).

To fulfil these conditions a letter used as a test a long distance from the eye must be larger and the constituent lines must be broader than in the case of a letter to be used nearer the eye. In Snellen's types the largest letter will subtend 5 minutes at the nodal point if it is 60 metres from the eye. Those in the subsequent lines will subtend 5 minutes if they are 36, 24, 18, 12, 9 and 6 metres from the eye. Sometimes smaller letters correspond-

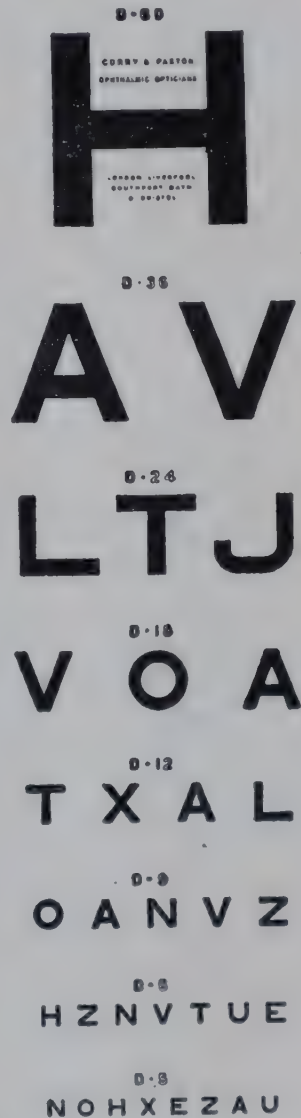


FIG. 127. Snellen's Distant Test Types (reduced). The lines, from above downwards, should be read at 60, 36, 24, 18, 12, 9, 6 and 5 metres respectively, i.e., at these distances the letters subtend a visual angle of 5'.

ing to 5 and 4 metres are used. A person with average acuity of vision ought therefore to be able to read the top letter at 60 metres, the **second** line at 36, the third at 24, and so on.

For convenience the patient is kept at a fixed distance from the types. This distance should never be less than 5 and preferably 6 metres. At such a distance the divergence of the rays in the small bundle which enters the pupil is so slight that the rays can be considered parallel and accommodation is thus eliminated.

A normal patient 6 metres from the types ought to be able to read every letter from the top to the end of the 6 metre line ; many people can read more in a good light. If the patient can only read the 18 metre line, his distant vision is obviously defective. The numerical convention which is used to record this is a fraction in which the numerator is the distance at which he is from the types, and the denominator is the

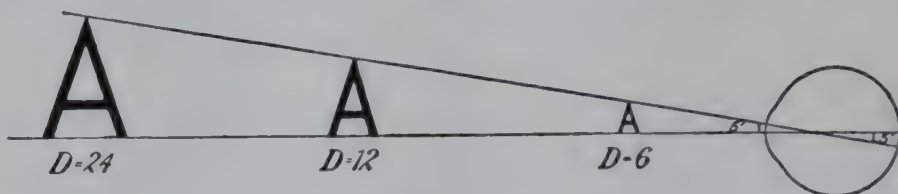


FIG. 128.

distance at which a person with normal vision ought to be able to read the last line which he succeeds in reading. The patient under consideration will therefore have his distant vision recorded thus : $V = 6/18$. The normal patient's vision will be $V = 6/6$.

These fractions should not be reduced, because they are conventions giving an accurate numerical estimate under special conditions. In its original form it indicates the actual types used and the distance away from the test-types.

In the United States of America the metric system is not usually employed (6 metres = 20 feet) : vision of 6/6 is therefore 20/20 ; of 6/60, 20/200 ; of 1/60, 3/200 (approx.), etc. (see Appendix II).

Other notations than the original suggestion of Snellen are widely used. On the continent of Europe many employ Monoyer's scale in arithmetical progression wherein the relative sizes of the test types are 10/10, 10/9, 10/8 . . . giving a relative visual acuity of 1.0, 0.9, 0.8 . . . 0.1.

The amount of illumination on the test card has a considerable influence on normal visual acuity. It has been found that the acuity rises rapidly as the illumination is increased from zero up to 5-10 foot candles ; and more slowly up to 1,000 or more ft. cs. (Chap. 37). The illumination of the test card should never be allowed to fall below 20 ft. cs., and to allow for the deterioration of lamps with use it would be advantageous if a standard of 100 ft. cs. were used.

If the patient cannot read the largest letter he is told to walk slowly towards the types. At a certain distance he may be able to see the top letter. He should then be moved back a little, since he may not have understood exactly where to look. In this manner the farthest point at which he can distinguish the top letter is determined. If this is 3 metres, the vision is recorded thus—

$V = 3/60$. If he is unable to see the top letter when close to it, he is asked to count the extended fingers of the surgeon's hand, held up at about 1 metre against a dark background ; this is recorded thus— $V = \text{fingers at 1 metre}$. If he cannot count fingers the surgeon's hand is moved in front of the eye ; if he can distinguish the movements the vision is recorded as $V = \text{hand movements}$. If he is unable to see these he is taken into the dark room and a light is concentrated on his eye and he is asked to say when the light is on the eye and when it is off. If he succeeds in doing this, $V = \text{p.l. (perception of light)}$ and he may be able to give some indication of the direction from which the light is coming (projection of light, good or bad). If he fails to see the light the vision is recorded as $V = \text{no p.l.}$

The measurement gives the visual acuity of the eye unaided by lenses. It is necessary in all cases, however, to determine the function of the macula in the best optical conditions, and for this purpose the refraction of the eye, including the manifest hypermetropia must be determined, and the visual acuity taken again in the same way with the correcting glasses in place. If, for example, there are two dioptries of hypermetropia, *corrected visual acuity* is then written : $V = 6/12 + 2D = 6/6$.

The ordinary test-types cannot be used with young children. Simple pictures constructed on Snellen's principles may be used. A very effective test is the "E-test" wherein the examiner holds in various positions cards whereon is printed the letter E in various sizes, and the child standing 6 metres away, if the matter is treated as a game, will readily respond on request by indicating the direction of the letter with his hand or by holding a similar card in the same positions so long as he sees it.

An objective measure of the visual acuity may be made by utilizing the phenomenon of *optico-kinetic nystagmus*. If a white drum with vertical black stripes is rotated before the eyes, these follow a stripe with a slow motion and then as it disappears, switch suddenly back to pick up a new stripe. This is an automatic reflex persisting so long as the individual stripes are seen, and by varying their breadth or the distance of the patient from the drum, an assessment of the acuity can be made, particularly in unco-operative or malingering patients. It is interesting that in cases of hemianopia the response is absent in lesions of the parietal lobe.

The Field of Vision. There are several methods of testing the field of vision.

(1) A rough, but very useful, method is the *confrontation test*, which should be applied in every case, at any rate if there is the slightest suspicion of defect, as follows :

The surgeon stands facing the patient at a distance of about 2 feet. The patient covers his left eye with the palm of his hand. He is told to look straight into the surgeon's left eye. The surgeon closes his right eye. He then moves his hand in from the periphery towards the common line of vision of the patient's right and his own left eye, keeping his hand in the plane half-way between the patient and himself. Directly he sees it himself the patient ought to say that he also sees it. The movements of the hand are repeated in various parts of the field—above, below, to the right, to the left, and so on.

This method is extremely simple, rapidly applied, and an excellent test. It will be seen that the surgeon tests the range of the patient's field by that of his own, which may be considered normal; moreover, he is continually watching the patient's eye, so that he can at once observe any deflection from the point of fixation. A hemianopic defect can easily be detected if the surgeon extends each hand to either side and asks the patient how many hands he sees.

If any defect is indicated by these methods or is suspected from other features of the case it must be accurately mapped out and recorded with the perimeter.

(2) *The Perimeter.* The perimeter consists essentially of a half sphere within which a spot of light can be moved (e.g., Goldmann's perimeter) or a rotatable arc, capable of being revolved round a pivot and along which a test-object can be moved (e.g., Lister's perimeter, Fig. 129). The former is the more accurate but is elaborate. The chart, which has concentric circles marked upon it corresponding to degrees on the arc, is under the surgeon's control at the back of the perimeter. In self-registering perimeters, which are almost invariably used, the readings are recorded by perforations with a sharp point.

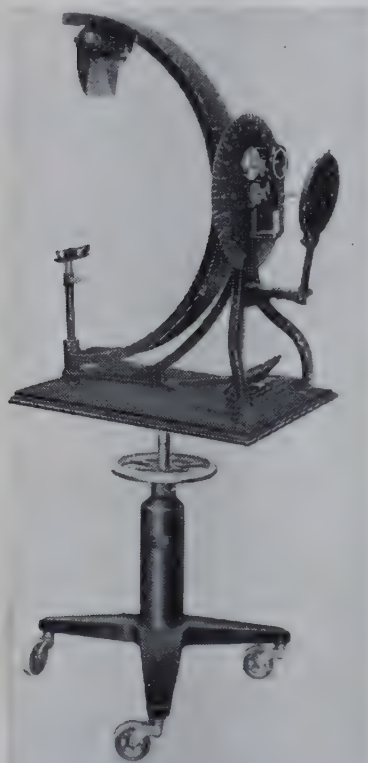


FIG. 129. Lister perimeter and scotometer.

The details of taking a perimetric chart can only be taught by actual demonstration. It will suffice to emphasize here the chief procedures to be followed in order that accuracy may be attained.

The patient is seated with his chin upon the chin-rest and the face vertical; one eye is covered. The other eye, situated at the centre of the arc, fixes the white dot around which the arc revolves.

The field should first be taken with a white object 5 mm. in diameter. At least eight meridians must be investigated, preferably sixteen, and the object should be carried up to the fixation point as there may be areas inside the limits of the field which are blind (*absolute scotomata*). These should be mapped out with the same accuracy as the limits of the field, and the plotting should always be from a blind to a seeing area. If the scotomata are small the limits should now be determined with a small object (1 or 3 mm. diameter). The size of the test object and its distance from the patient's eye are usually recorded by a convention similar to the mode of recording visual acuity, e.g., 5/330, both measurements being expressed in millimetres. With small objects *relative scotomata* can be found which are not demonstrable with large objects.

Perimetry is a relatively rough test and purely subjective. Every student should have his own field taken : he will then appreciate the difficulties which patients experience. The normal physiological response to an object in the peripheral field is to turn the eyes towards it. In charting the field of vision this normal response must be suppressed, fixation being rigidly maintained while attention is directed to an object at the periphery. Hence the first fields taken should be regarded with suspicion, especially in the case of dull or neurotic patients. The most variable factor is the illumination, and sufficient attention is not usually paid to this point. With good illumination an object subtending a visual angle of 0.5° will give the full normal field for white. The 5 mm. object used at the ordinary distance of 33 cm. ($5/330$), corresponds to a visual angle of approximately 1° .

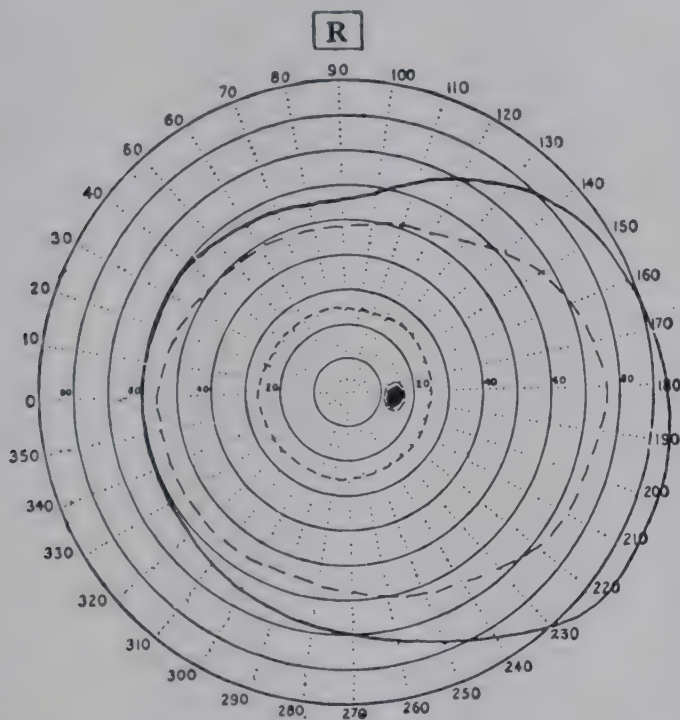


FIG. 130. The right visual field for a white object determined on the perimeter. ($5/330$, $1/330$, $1/2,000$ in good daylight.)

The extent of the normal field, with a 5 mm. object in good illumination, is shown in the accompanying chart (Fig. 130). The peculiar shape is essentially due to the shape of the sensitive area of the retina as projected outwards, modified by interference caused by the nose and the brows ; this complication can be eliminated if, when the field of the right eye is being taken, the head is turned somewhat to the left, and *vice versa*. It is seen that the field for white extends upwards 60° , outwards rather more than 90° , downwards 70° , and inwards 60° . The size varies with the illumination, the size of the test object, the contrast of the test object with the background, and the state of adaptation of the eye.

If the charts of the two eyes are superimposed there will be a large central area which is common to both eyes : this is the *field of binocular vision*.

Having mapped out the field for white the process may be repeated with coloured objects of similar size. The limit of the field for a colour is the point at which, passing from the periphery to the centre, the colour first becomes evident; peripheral to this limit the object is perceptible but appears grey in ordinary illumination. The exact limit is difficult to determine, for most colours appear to change in hue and saturation as the object passes from the fixation point towards the periphery. Red or green should be used first, then blue or yellow. In ordinary conditions, the blue field is largest, slightly smaller than the white: then follow the yellow, red and green, in the order named. There is a particular purplish-red and a particular bluish-green ($490\text{ m}\mu$) which have the same field; similarly a particular blue ($460\text{ m}\mu$) and a particular yellow ($570\text{ m}\mu$). These pairs of colours are complementary, i.e., a mixture of the red and the green, or of the blue and the yellow, produces white.

The field for blue and yellow is roughly 10° less in each direction than that for white, that for red and green another 10° less. The limits of the colour fields vary not only with the intensity of the light, but also with saturation of the colour, and above all with the size of the object. If these are sufficiently great, colours may be recognized almost, if not quite, at the periphery. Deductions made from variations in the colour fields are particularly unreliable.

(3) For more accurate investigation of details *campimetry* must be employed, but it is applicable only to the central and paracentral areas. It consists of placing the patient 2 metres from the centre of a large black screen, 2 metres or more in diameter (Bjerrum's screen). He fixes a spot in the centre of the screen and small white targets in the form of discs, 1 mm. to 10 mm. in diameter, attached to a long black rod are brought in from the periphery on a level with

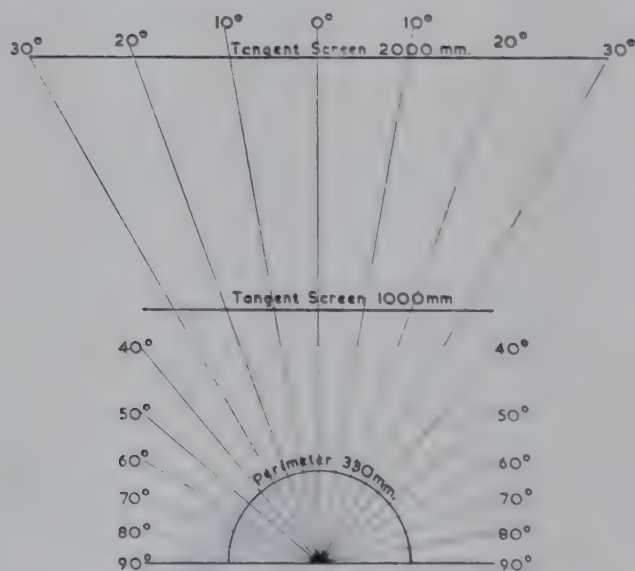


FIG. 131. Diagram of the right field, showing the relationship of the retina to the degrees of the perimetric arc, and the relative value of the latter when projected on a tangent scale.

the screen. A grey screen with a spot of light the size of which can be controlled may be used in a similar fashion; this method has the advantage of eliminating the distraction caused by the rod. At this distance a 3 mm. object subtends a visual angle of about 5 minutes. It will be noticed that since the angles are projected onto a flat surface, tangents are recorded, not angles themselves as with the arc (Fig. 131). Hence only a small area can be investigated, and the distortion must be taken into account. Some points of diagnostic importance which cannot be elicited by the perimeter can be brought out by this method. Various *scotometers* have been devised on the principle of Bjerrum's screen.

The Light Sense should never be measured until the patient has become thoroughly dark-adapted by remaining at least twenty minutes in a dark room. For this measurement instruments called *photometers* (or *adaptometers*) have been used, but none of them is of easy clinical application. There are individual differences in the rate of development of dark adaptation and facility of behaviour under low illumination which must be considered normal, but the rate of dark adaptation may be prolonged in pathological conditions such as pigmentary dystrophy of the retina, vitamin A deficiency or glaucoma. Such investigations, however, do not lend themselves readily to routine clinical investigation.

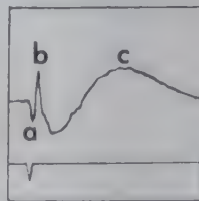
The Colour Sense requires elaborate apparatus for its scientific investigation. The methods used will be discussed later (p. 360).

It is frequently advisable, however, to investigate the central part of the field for red and green, since conditions are not uncommon (such as tobacco amblyopia and retrobulbar neuritis) in which these colours are not recognized by central vision (*central relative scotomata*). In such a test it is sufficient to use perimetric targets of the appropriate colour (5 mm. in diameter). It will be found that blue and yellow will frequently be recognized as such but not red and green.

The Objective Examination of Retinal Function

The visual function can be explored objectively by *electroretinography* (ERG) whereby changes induced by the stimulation of light in the resting

FIG. 132. Typical electroretinogram showing *a*, *b* and *c* waves. The dip in the lower line indicates the point of stimulation.



potential of the eye are measured. In the normal dark-adapted eye, after a fleeting early receptor potential, three components are seen (Fig. 132): a negative *a*-wave, possibly representing the activity of

the rods and cones, a positive (composite) *b*-wave arising in the inner retinal layers, and, with strong stimuli, a secondary rise in potential, the *c*-wave, related not to visual processes but to the retinal metabolism, associated particularly with the pigmentary epithelium. Clinically the simplest technique investigates the dark-adapted eye wherein a minute *a*-wave is followed by the positive *b*-wave (Fig. 132). It is measured in dark adaptation with the active electrode incorporated into a contact lens and the reference electrode attached to the forehead so that a monopolar recording is obtained of the electric potentials picked up from the corneal surface. The response is *extinguished* when there is complete failure in the function of the rods and cones (pigmentary retinal dystrophy, complete occlusion of the retinal artery, complete retinal detachment, advanced siderosis, etc.). It is *subnormal* in those conditions wherein a large area of the retina does not function; and *negative* in gross disturbances of the retinal circulation (Fig. 133).

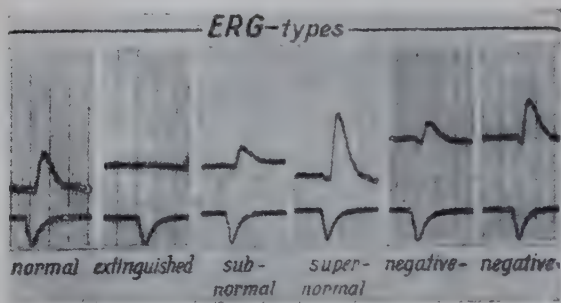


FIG. 133. Types of electroretinogram. Upper curve, ERG. Lower curve, photometric record of light-flash.

The electroretinogram essentially gives an indication of the activity of the periphery of the retina, that is, of the rods and their immediate connections. The photopic electroretinogram makes use of the critical fusion frequency of flicker, but so far has few clinical applications. By using a red filter and sharply focused light, however, an electroretinogram can be obtained from the fovea by the use of averaging techniques; in this way a macular degeneration can be diagnosed in cases of cataract.

In the related technique of *electro-oculography* (EOG), changes in the resting current when the eyes are moved laterally are picked up by electrodes placed at the inner and outer canthi. Changes in the potential thus obtained with changes of illumination are indicative of the activity of the pigmentary epithelium and the outer segments of the visual receptors and are diminished or absent in retinal dystrophies and degenerations often before visual symptoms are evident. The technique is thus of value in diagnosing objectively such diseases in their early stages or in cases wherein the fundus cannot be clearly seen.

CHAPTER 14

GENERAL THERAPEUTICS

THERAPEUTIC substances may be introduced into the eye by five methods—by instillation into the conjunctival sac, by subconjunctival injection, by iontophoresis, by systemic administration and by direct injection into the globe itself.

1. On *instillation* in the form of drops or ointments drugs enter the eye largely through the cornea and the readiness with which solutes instilled into the conjunctival sac can be detected in the aqueous or (as with atropine) can be evident as acting on the intra-ocular tissues shows the extent of their permeability. The cornea is freely permeable to water, but, largely through the action of the epithelium and to a less extent of the endothelium, it offers considerable resistance to the passage of electrolytes, while the passage of large colloidal molecules is barred. The stroma, like the sclera, is permeable to all water-soluble substances, offering a resistance little greater than an isotonic solution of sodium chloride. In general terms the passage of drugs through the epithelium is determined by the factors controlling the penetration of drugs into other cells, two of the most important of which are fat-solubility and the degree of dissociation of the electrolyte.

Since the epithelium forms the main barrier, permeability is much increased if the epithelium is damaged or abraded or if its vitality is impaired by a local anæsthetic; this is well demonstrated by the staining of the tissue by fluorescein. Theoretically the permeability to water-soluble substances is also increased by detergents ("wetting agents") which, possessing a lipophilic grouping, lower surface tension. Such substances, however, are often toxic to the corneal epithelium and should be used, if at all, sparingly, and never in cases of corneal disease.

2. By *subconjunctival injection* a much wider range of substances can be introduced into the eye, for the sclera allows the free and indiscriminate transit of molecules of considerable size. Thus penicillin which does not penetrate the cornea enters the eye freely by this route. Since the conjunctiva has a less rigorous semi-permeability than the corneal epithelium, a less effective but useful method of introducing drugs through the sclera is by leaving a pledget of cotton wool soaked in the drug (a tampon) in the fornix. Penicillin can be introduced into the eye in this way in considerable quantity. Ointments probably enter the eye in this way, as well as through the cornea.

3. By *iontophoresis* an electrolyte is driven into the eye with the passage of a galvanic current. The permeability of the cornea can thus be considerably increased, but in many cases the effect is probably largely due to epithelial damage.

4. The *systemic route*, by which drugs can be given by mouth or injection, has its obvious limitations because of the impermeability of the blood-aqueous barrier. No passage is allowed to large-sized mole-

cules (such as the arsenic compounds or penicillin) and when molecular size is at the border-line, lipid-solubility is the most important asset. Thus the rate of entry of sulphanilamide (a lipid-soluble substance) is sixteen times that of sucrose although their molecular sizes are comparable; the transformation of urea (non-lipoid-soluble) into the larger-molecule but lipid-soluble thiourea increases its ease of passage five times, while among the common antibiotic drugs, the lipid-soluble chloramphenicol enters the eye the most freely.

5. *Injection into the eye*, either into the anterior chamber or the vitreous, is reserved for desperate cases; it is employed, for example, to flood the ocular tissues with penicillin in acute infective conditions or with cortisone in sympathetic ophthalmitis. The technique employed is to introduce a sharp needle, most easily done through a preliminary valvular puncture with a sharp Graefe knife (p. 411), to withdraw a small quantity of fluid and, after rapidly changing the syringe, to introduce a corresponding quantity of solution into the globe. When the globe is already opened operatively, of course, such a technique is easy.

CHEMOTHERAPY AND THE ANTIBIOTICS

The greatest revolution which has occurred in therapeutics during this century, not only in ophthalmology but in the whole of medicine, has been the discovery and exploitation of drugs which have the property of doing more harm to invading organisms than to the tissues of the body. Antiseptics act by killing both indiscriminately. Chemotherapeutic and antibiotic drugs, however, are bacteriostatic rather than bactericidal, acting probably by competing for the raw materials necessary for the existence of the organisms. The result is that since the organisms are inhibited from growing and multiplying, the natural defences of the body can deal with those already present. As soon as the influence of the drug is withdrawn, the remaining organisms can resume growth and multiplication, so that the rationale of treatment is to keep the drug continuously in contact with the infected tissue until the infection is overcome. Since these drugs are rapidly excreted from the body or diffuse from any site of local application, repeated or continuous administration during this crucial period is necessary.

The essential value of all these drugs is in the treatment of acute infections; in chronic infections they are relatively ineffective or relapses follow their use. This applies particularly to the more chronic types of intra-ocular disease which show little or no response, a circumstance perhaps due to the allergic rather than the directly infective nature of many of these conditions.

Chemotherapeutic drugs have been known since the treatment of syphilis by arsenical compounds was introduced. For the common ophthalmological infections, however, the most important compounds are the sulphonamides, the sulphones, para-amino-salicylic acid and Isoniazid.

The Sulphonamides. Several drugs belonging to this group are in general use, the newer compounds having the general advantage of being less liable to cause toxic effects than the original sulphanilamide (prontosil). The more commonly used are sulphadiazine, sulphadimidine, sulphamerazine and the long-acting compounds such as sulphamethoxypyridazine or sulphaphenazole.

The sulphonamide drugs have a wide range of usefulness which embraces a number of ocular infections. They are peculiarly effective against hæmolytic streptococci, gonococci, pneumococci, staphylococcus aureus, meningococci, diplobacilli, the influenza bacillus, coliform bacilli, Friedländer's bacillus and the gas-gangrene organisms. Their activity varies considerably from the great effectivity shown against the beta-hæmolytic streptococcus to their relatively low activity against the staphylococcus. Tuberculous, leprosy, syphilitic, brucellar or tularemia infections show no response, nor do those due to non-hæmolytic or anaerobic streptococci. None of the typical viruses is affected, but three larger *Bedsonia* of ophthalmic interest are sulphonamide-sensitive—those of trachoma, inclusion blennorrhœa and lymphogranuloma venereum. In all cases, however, resistant strains of organisms are encountered.

The sulphonamides may be administered either systemically or locally. Being lipoid-soluble, they cross the blood-aqueous barrier with relative freedom so that systemic administration is effective in acute ocular diseases, as well as in similar infections of the conjunctiva, lids and orbit.

In their *systemic administration* an attempt should be made to obtain the maximum concentration in the blood of 100 mg. per 100 ml. over a period sufficient to allow the infection to be overcome. Theoretically the dosage should be calculated for each patient on a basis of body-weight, but in practice in the average adult an initial dose of 2.0 G. followed by a 6-hourly maintenance dose of 1.0 G. is suitable. For a newly born infant doses 1/10 of this are indicated. The long-acting sulphonamides are given in an average dose of 1.0 G. initially followed by 0.5 G. daily or 1.0 G. every second day.

The *local use* of sulphonamides for conjunctival or corneal infections has largely been replaced by that of antibiotics; nevertheless they have some application. The most effective for general application because of its complete lack of irritability is sodium sulphacetamide (Albucid) which may be employed as a solution in drops (30 per cent.) or as an ointment (5 per cent.).

The Sulphones were elaborated with a view to attacking the tubercle bacillus but are now widely used with success in the treatment of leprosy.

Para-amino-salicylic acid (PAS), given systemically by mouth in four daily doses of 3 G. each, or alternatively, *isonicotinic acid hydrazide* (Isoniazid) in two daily doses of 2.5 mg. per kg. body-weight, forms a much more effective combination with streptomycin than the sulphones against tuberculosis; each appears to reinforce the action of streptomycin and at the same time appears to preserve the sensitivity of the bacillus to this drug. The combination is effective in treating both intra- and extra-ocular tuberculosis of an infective (not allergic) nature.

The antibiotics are a class of substances derived from fungi or other bacteria ; their action depends on the inhibitory effect which one organism exerts on another. This "antibiosis" has long been known, but the discovery of the prototype of such drugs—penicillin—only dates from the work of Fleming in 1929. In the last few years many such drugs have been exploited and used because of their activity against different organisms, the most important of which are penicillin, streptomycin, chloramphenicol and the various tetracyclines. In general, penicillin is effective against Gram-positive organisms and certain spirochætes ; streptomycin against Gram-negative organisms and certain acid-fast species, while the "broad spectrum" antibiotics—chloramphenicol, tetracycline, chlortetracycline, oxytetracycline, neomycin, soframycin and others—are clinically effective against both Gram-positive and Gram-negative organisms as well as rickettsiæ, the Bedsonia and certain spirochætes and protozoa. With the exception of those due to the *H. ægyptius* and the viruses (epidemic kerato-conjunctivitis, herpes, etc.), all the common acute infections of the outer eye are susceptible to one or other of the antibiotics. Many fungi are sensitive to nystatin, amphotericin or trichomycin. It is to be remembered that all these substances are inactivated by the heavy metals ; lotions containing zinc or mercury should not therefore be prescribed along with them.

Penicillin, obtained from the mould *Penicillium notatum*, is the most generally useful of these drugs, partly because of its ready availability and cheapness, partly because of the wide range of organisms against which it is effective and partly because, apart from the hypersensitivity which a considerable number of people exhibit towards it, it is relatively non-toxic. Some of its recently produced relations are more widely useful. In general it is an extraordinarily effective bacteriostatic agent to which Gram-positive organisms and Gram-negative cocci are sensitive ; the Gram-negative bacilli as a class are relatively insensitive to it. It is, however, a large-moleculed substance and, unlike the sulphonamides, cannot diffuse into the eye in effective quantity unless administered in very high concentration. While the sulphonamides are therefore indicated in intra-ocular infections, penicillin is most generally useful in extra-ocular infections. In deep-seated inflammations of the orbit or lids it is administered parenterally ; in superficial inflammations of the conjunctiva and cornea it is administered locally as drops, ointment or powder. In intra-ocular infections it is given as injections subconjunctivally or (occasionally) directly into the eye.

Systemic Administration. To maintain an effective concentration of penicillin in the tissues a daily dose of the order of 300,000 to 1,000,000 units of one of the less soluble and more slowly absorbed preparations of penicillin should be administered for some days (such as Distaquaine). Preparations are also available for the administration of penicillin by mouth in capsule or tablet form (Distaquaine, Penbritin) ; their troublesome effects on the mucosa of the mouth and bowel should be counteracted by the administration of large doses of vitamin B.

Local Administration. Penicillin may be given in the form of drops,

ointment or powder or as subconjunctival injections, the pure sodium salt being always employed. Drops should contain from 1,000 to 10,000 units per ml. and should be instilled into the eye at frequent intervals, depending on the acuteness of the infection. In the early stages of an acute infection, intervals varying from a minute to a quarter of an hour may be indicated; in less acute infections intervals of two to three hours will be sufficient. An ointment may be made on a basis of soft yellow paraffin at a strength of 5,000 units or more per G. (for details see App. I). The ointment produces a less potent but more prolonged effect and is more liable to cause an irritation of the lids than the drops.

Powder. For use in powder form dry calcium penicillin is diluted with one of the sulphonamide powders (sulphathiazole) to give a concentration of from 1,000 to 5,000 units per G. Such a powder can be used in the treatment of corneal ulcers and in the treatment of an infected socket or dusted into an open wound of the lids or orbit.

Intra-ocular Administration. The most convenient method of administration is by subconjunctival injections of 500,000 units or more with adrenaline repeated every three hours (see App. I). In desperate cases of intra-ocular suppuration the solution may be injected directly into the anterior chamber or the vitreous: for this purpose the drug must be absolutely pure lest an intense and sometimes destructive reaction follow.

Streptomycin, a derivative of an actinomyces, has a considerable antibacterial spectrum. It is effective against the staphylococcus, streptococcus and other Gram-positive bacteria, actinomyces and *M. tuberculosis*, as well as against a large number of Gram-negative bacilli—*E. coli*, *P. tularensis*, *Br. abortus*, *Pr. vulgaris*, *H. pertussis*, *H. influenzae* and many others. It is thus effective against many organisms which are penicillin-resistant, the most important of which is the tubercle bacillus. The drug is, however, toxic after prolonged administration, the most important complication, fortunately not common, being an affection of the eighth nerve causing giddiness, deafness and sometimes nystagmus. Moreover, organisms frequently tend to acquire a resistance to the drug. For these reasons in ophthalmology its systemic use is usually confined to the treatment of tuberculosis when it is most effectively used in combination with PAS (*q.v.*).

Tetracycline (*Achromycin*), *chlortetracycline* (*Aureomycin*), *oxytetracycline* (*Terramycin*) and *Neomycin* are "broad-spectrum" antibiotics with a considerable antibacterial action against both Gram-positive and Gram-negative organisms, as well as some fungi, rickettsiae and the *Bedsonia*; the latter group includes the infective agent of trachoma. Their ability to penetrate the ocular tissues, however, either from the conjunctival sac or after systemic administration, is small—less, indeed, than that of penicillin. They are therefore essentially employed in the form of drops or ointments for superficial ocular infections; they are too irritative for subconjunctival injection.

Chloromycetin, originally derived from a streptomyces and now synthesized as *chloramphenicol*, has a somewhat similar antibacterial spectrum, being also effective against the *Bedsonia*. The molecule is

relatively small and lipoid-soluble, so that on systemic administration it enters the eye in therapeutic concentrations. Its effects, however, on intra-ocular inflammations, are usually not dramatic.

The *Polymyxins*, isolated from different strains of the *B. polymyxa*, are potent antibiotics against Gram-negative bacteria. In view of the fact that most other antibiotics do not affect the organism, they are useful against extra- or intra-ocular infections of *Ps. pyocyanea*, administered as drops in the first case or as subconjunctival injections in the second.

Soframycin is highly effective against Gram-positive cocci and Gram-negative bacilli including *Ps. pyocyanea*. Moreover, being non-irritable, it is suitable for subconjunctival injection which increases its local action manifold.

In general it may be said that the infections susceptible to the sulphonamides are also susceptible to antibiotics ; and of the two the greater rapidity of action and the less toxicity make treatment with antibiotics the method of choice. Moreover, the inactivation of the sulphonamides by pus lowers their applicability considerably in acute infections of the outer eye ; while, on the contrary, the ready permeability of these drugs through the blood-aqueous barrier and the difficulty experienced by most antibiotics in penetrating into the inner eye, make the former the method of choice for treating those intra-ocular infections amenable to chemotherapeusis unless the infection is of sufficient severity to merit subconjunctival injections. In ophthalmology, therefore, where acute bacterial infections are concerned, sulphonamides find their greatest value in systemic administration, the antibiotic substances by topical administration. If the condition warrants it, of course, there is no contra-indication to the employment of both ; in fact, on occasion, such treatment may have synergic advantages as in the combination of streptomycin and Isoniazid or para-amino-salicylic acid.

On the whole, however, probably because so many extra- and intra-ocular inflammations are not directly infective in nature, the revolution which these new methods of therapeutics have brought to ophthalmology is not so dramatic as has been experienced elsewhere in medicine. The outstandingly dramatic cures obtained in such conditions as were endowed a decade ago with the utmost gravity, as purulent gonococcal ophthalmia, hypopyon ulcer or exogenous intra-ocular infections, are by no means paralleled in the clinical behaviour of the average case of endogenous intra-ocular inflammation. Moreover, even in the most favourable type of case, the delicacy of the ocular tissues and their inability to regain functional integrity after damage of any marked severity, make antibacterial treatment of comparatively little value unless it is instituted early. Finally, it should always be remembered that in all cases these drugs are not panaceas, appropriate ancillary

treatment is necessary, and their use even with lavish prodigality will not compensate for the lack of good surgery or provide a cover for inadequate therapeutics.

Antiviral substances have proved more elusive. The Bedsonia which have a partial extracellular existence and are not true viruses (trachoma, inclusion conjunctivitis) are susceptible to antibiotics, but the small strictly intracellular viruses which utilize the cells of the host for their metabolic needs are immune. Antiviral drugs must therefore inhibit viral replication. One such substance, *interferon*, damps down the oxidative processes necessary for viral synthesis and is effective against the pox viruses and others. Another, 5-iodo-2-deoxyuridine (IDU), inhibits the synthesis of DNA and thus, on topical application, prevents the replication of such viruses as that of herpes. A somewhat similar action is attained by cytosine arabinoside HCl (CA) which blocks the synthesis of nucleic acids. The use of these agents is still in the early stage, but it is probable that a completely new field of therapeutics is about to be opened up in this way.

HORMONE THERAPY BY THE CORTICOSTEROIDS

Of recent years much interest has been excited by the therapeutic effect of certain steroids elaborated by the adrenal cortex, the first of which was cortisone. This hormone can be used effectively in ophthalmological conditions; indeed, the eye is a very suitable organ for its exploitation for the undoubted toxic effects resulting from its systemic administration can be eliminated by its topical use in ophthalmological practice. Greater potency for topical application is obtained by the related compound, hydrocortisone; while the newer synthetic drugs such as Prednisone, Prednisolone, Dexamethasone and others, are often preferable for systemic administration since they are less liable to excite the unfortunate side-effects associated with cortisone. They are not, however, without their dangers, among which the possibility of the causation of glaucoma in genetically susceptible persons should be remembered. Clinically the same therapeutic reaction can be obtained by the administration of the adrenocorticotrophic hormone of the hypophysis (ACTH); this preparation excites the production of cortisone by the adrenals and is thus only effective on systemic administration.

The general clinical effect in ocular disease of these hormones is a temporary blockage of the exudative phases of inflammation and an inhibition of fibroblastic formation in the process of tissue-repair, whether the cause of the disease is bacterial, anaphylactic, allergic or traumatic. In acute inflammations capillary permeability is decreased and the cellular exudation reduced; while in the stage of healing the formation of granulation tissue, new vessels and fibrosis is reduced. It is, however, of the utmost importance to

remember that cortisone does not affect the cause of any disease but merely provides mesenchymal tissues with a temporary protection against an irritant, organismal or otherwise. The tissue-cells become resistant to an injury and are rendered able to function normally in an environment which has become grossly abnormal. The effect is thus limited to the blocking of the pathological evidences of inflammation so long as the administration of the hormone is continued ; on its withdrawal the disease forthwith resumes its natural course. It follows that cortisone has its greatest effect in the control of acute disease ; it is completely ineffective in the removal of structural damage caused by old or long-standing inflammation, nor is it of any value in the treatment of degenerative conditions.

Cortisone is thus in no sense curative. Its essential function is to hold the acute phases of inflammation in check while cure is obtained by other methods. Thus in infective conditions, cure in the true sense can only be obtained by killing off the invading organisms, as by an antibiotic. In an organ composed of tissues so delicate as the eye, however, this inhibition of the inflammatory reaction is often of the first importance, for damage of a degree which may be tolerated by other tissues may well have a disastrous effect on vision ; indeed, if this restraint can be maintained until the organism is dealt with by other means, in the end the eye may escape all permanent damage. On the other hand, if an infection is not otherwise eliminated, the disease resumes its course on the cessation of cortisone treatment and apart from a temporary respite, little good results. In such cases, indeed, the treatment may be a disadvantage, for the tissue-response to injury is essentially protective in its function and its inhibition may not be without danger. The spread of tuberculosis, for example, may be facilitated by the suppression of fibrosis while the organism still lives ; while if cortisone is given in an acute infection of the cornea the causal organisms of which are not sensitive to drugs (as in the case of many viruses), since the normal inflammatory response is inhibited, although the eye may seem quiet the infection runs on apace, often with disastrous consequences. The ideal therapeusis is therefore the control of the deleterious aspects of the inflammatory response by cortisone but only if the infective or other cause can be eliminated by other means.

In ophthalmology these steroids may be administered locally or systemically. *Local administration* is by drops, ointment or injection. Drops are used every two or three hours as a 1 in 4 dilution of the standard preparations in saline or a buffered phosphate vehicle. The ointment (25 mg./G.) is equally well absorbed into the eye and has a longer action, being particularly useful for administration at night. Subconjunctival injections have a more powerful effect lasting two or three days ; 0.2 to 0.4 ml. are injected after local analgesia behind the limbus. Retrobulbar injections have been tried with a view to controlling inflammations of the posterior segment of the eye,

but they are relatively ineffective. Injections into the anterior chamber may be used in desperate cases.

The steroids may also be administered *systemically*, either by mouth or subcutaneous injection, but this requires careful clinical control. The main value of such treatment is in acute inflammatory disease of the posterior segment of the globe which is not readily affected by local therapy. In severe and acute cases the most dramatic effect is attained by the intravenous injection of ACTH. It should be remembered that after the prolonged administration of certain synthetic steroids (prednisone, dexamethasone, triamcinolone) cataractous changes may occasionally occur, and (in susceptible patients) simple glaucoma.

In ophthalmology local therapy by this group of substances has been found beneficial in allergic and certain infective conditions, particularly early deep keratitis (including zoster and other viral diseases), syphilitic interstitial keratitis, phlyctenular and rosacea keratitis, spring catarrh, episcleritis, the acute and subacute phases of iridocyclitis and particularly sympathetic ophthalmitis. Systemic therapy is of value in acute or subacute generalized uveitis or focal choroiditis.

In all chronic inflammations their administration gives temporary or irregular results; in degenerative conditions they are without effect. In no disease are they curative, and if they are relied upon as the only method of therapy, the factor dominating the prognosis in almost every case is the occurrence of relapses on their cessation unless the malady is eradicable or self-limiting.

ARTIFICIAL FEVER (SHOCK) THERAPY

In certain aspects the response to cortisone resembles that to fever therapy by "protein shock" and, indeed, there is evidence that the latter method of treatment may act largely by stimulating the adrenal cortex to secrete steroids. The technique has now been largely abandoned in favour of corticosteroids but it may be of value in producing a beneficial effect in certain subacute or chronic ocular inflammations, particularly of the cornea or uveal tract. Like that of cortisone the effect is temporary, but not infrequently the amelioration allows other methods of treatment, hitherto without effect, to control the situation.

The treatment is given by the injection of foreign protein. The general reaction is at first characterized by a low temperature with rigor, slow pulse and nausea; this is followed by a high temperature, rapid pulse and leucocytosis.

The most useful method is the intravenous injection of typhoid vaccine, an initial dose of 10 million organisms being followed, depending on the severity of the reaction, by doses up to 100 million. In each case the succeeding injection is best given some 24 hours after the temperature has subsided. This has now largely replaced the initial technique of the intramuscular injection of milk.

TISSUE THERAPY has been advocated, initially by Filatov, with a view to the stimulation of tissues by the release of "biogenic substances" of an unknown nature. An apparent example of this is the clearance of surrounding opacities after the insertion of a corneal

graft. Whatever the theoretical basis for the concept, and it is undoubtedly obscure and questionable, the injection of a vast number of animal and vegetable products has been advocated in most of the types of degenerative ocular diseases that respond to no method of therapy—myopic chorio-retinal atrophy, pigmentary retinaldystrophy, optic atrophy, and so on. The most usual method practised is the intramuscular injection of placental extract or the subconjunctival implantation of placental tissue ; but most authorities agree that, apart from the psychological effect, the results are negligible.

SECTION IV

DISEASES OF THE EYE

CHAPTER 15

DISEASES OF THE CONJUNCTIVA

WITHIN the limits of health the conjunctiva shows very considerable variations in appearance at different ages and in people who follow various employments. The peculiarities of colour, vascularity or laxity which are consistent with health can be learnt only by repeated observations. This tissue is frequently the site of disease, for not only is it exposed to all types of exogenous irritants and infections and prone to allergic reactions thereto, but it often becomes involved in endogenous diseases and metabolic disturbances.

Anatomically the conjunctiva is divided into two portions, palpebral and bulbar; the folds uniting these parts are the fornices (Fig. 134). The palpebral conjunctiva is said to commence at the anterior margin of the edge of the lid, but from this point to the posterior margin of the edge (the intermarginal strip) and for about 2 mm. beyond (to the sulcus subtarsalis) there is a transitional zone covered with stratified epithelium with the characters of both skin and conjunctiva (Chap. 31). There are two layers of epithelium over the palpebral conjunctiva: from the fornices to the limbus the epithelium becomes gradually thicker, forming once more a stratified epithelium near the corneal margin. Below the epithelium is an adenoid layer, consisting of loose connective tissue containing leucocytes: below this is a fibrous layer, much denser and passing insensibly into the underlying tissues—lid or sclera. The palpebral conjunctiva is firmly adherent to the tarsus, while the bulbar portion is freely movable over the sclera except close to the cornea.

Bacteriology. The conjunctival sac is practically never free from organisms, but owing to its relatively low temperature due to exposure, evaporation of lacrimal fluid and moderate blood supply, bacteria do not readily propagate themselves. Moreover the tears are not a good

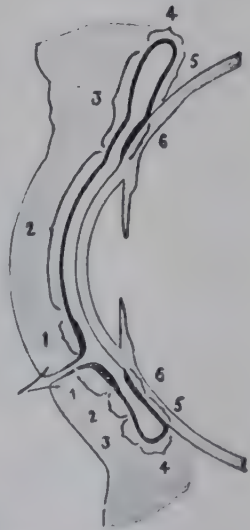


FIG. 134. The conjunctival areas. 1, marginal; 2, tarsal; 3, orbital; 4, fornix; 5, bulbar; 6, limbal.

culture medium, but although they contain a bacteriostatic enzyme, lysozyme, they cannot be regarded as actively bactericidal. Hence they act principally in a mechanical manner, washing away deleterious agents and their products. The bacterial content of the conjunctival sac is increased by bandaging owing to the arrest of movements of the lids and raising the temperature of the sac.

Most of the organisms normally present are non-pathogenic, but some of them are morphologically identical with pathogenic types. Diplococci indistinguishable from pneumococci are sometimes present and in general this organism (as well as *Ps. pyocyanea*) is among the most dangerous in ocular infections. The *Corynebacterium xerosis* is morphologically identical with the *C. diphtheriæ* and is frequently present in the normal conjunctival sac; it can only be distinguished by examination of cultures. Staphylococci are often found; they are relatively innocuous in the absence of other organisms but play an important part in mixed infections. Streptococci, *E. coli*, *B. proteus*, etc., are pathogenic, but rare. Other pathogenic organisms—gonococci, *H. ægyptius*, *Moraxella*—will be discussed later.

Viruses play a large part in conjunctival disease, as also do the Bedsonia. The most common viruses are that of herpes and the adenoviruses.

Hyperæmia of the conjunctiva may be transitory, or recurrent and chronic. The former is caused by temporary irritation, as by a foreign body in the conjunctival sac (which includes the surface of the cornea), concretions in the palpebral conjunctiva or in-growing lashes: in such a case the apparent secretion is almost wholly a reflex secretion of tears. Irritation limited to the lower fornix may be artificial in malingerers and psychopathic patients.

Recurrent or chronic congestion may be caused by conditions such as dusty, ill-ventilated rooms or exposure to strong light or heat, or refractive errors, but is often due to causes remote from the conjunctiva itself, such as errors of metabolism—gout, over-eating and drinking, and so on—or allergic conditions such as hay-fever; in the latter case there may be an excess of eosinophilic cells in the conjunctival secretion.

Simple hyperæmia of the type described causes a sense of discomfort, often described as tightness, grittiness, inability to keep the eyes open and tiredness. Bright light is resented, but there is seldom true photophobia. The conjunctiva often looks normal until the lower fornix is exposed, when it will be seen that the parts in contact are congested and sticky. An increased secretion of a watery nature is the rule and the presence of mucus suggests an infective condition. The discomfort frequently comes on only in the evening or after near work.

Whenever watering of the eyes is complained of, and whenever only one eye is congested or shows signs of conjunctivitis, the lacrimal passages must be investigated. Pressure with the finger backwards and inwards over the lacrimal sac may cause regurgitation of fluid—tears, mucus, or pus—showing

that the outflow into the nose is obstructed. If no regurgitation can be detected, the position of the lower punctum must be noted. It ought to be invisible until the lid is slightly everted.

The *treatment* of simple hyperæmia consists primarily in the removal of the cause whatever it may be—and some of them are readily overlooked. The irritation of strong light may be modified by the use of dark glasses. Local treatment consists in bathing the eyes frequently with a mild astringent lotion (zinc sulphate, etc., App. I). Transitory relief may be obtained by a drop of adrenaline solution (1 in 1,000) or in allergic cases a preparation such as antistine privine.

INFLAMMATION OF THE CONJUNCTIVA

Inflammation of the conjunctiva manifests itself in many grades and many types, but is usually of infective or allergic origin. It is always accompanied by hyperæmia and increased secretion. The hyperæmia varies in degree and in distribution : the secretion varies in nature and amount. The nature of the secretion is of diagnostic importance. It may be watery, due largely to an increased secretion of tears, or mucous, muco-purulent, or purulent, in which case the disease is usually due to a bacterial agency ; a serous secretion suggests a viral ætiology. Occasionally the exudation from the abnormally permeable capillaries is retained within the mucous membrane which becomes swollen and gelatinous in appearance particularly in the loosely attached areas of the bulbar conjunctiva and fornices ; this phenomenon is called *chemosis* and in severe cases the swollen membrane forms a wall around the cornea. The palpebral conjunctiva is little affected, but the tissues of the lid are often also oedematous, so that the lids are swollen.

In the diagnosis of conjunctivitis, bacteriological investigation should always be supplemented by histological examination of the secretion and of *scrapings of the epithelium* taken by a platinum loop and stained with Giemsa. Apart from the presence of bacteria or inclusion bodies in the cells in specific infections, the cytological picture provides useful information. A preponderance of polymorphonuclear cells indicates an acute bacterial infection ; mononuclears a viral or chronic bacterial infection ; multinucleated epithelial cells a viral infection ; plasma cells trachoma ; and eosinophils and basophil cells an allergic reaction.

Infective Types of Conjunctivitis

The chief forms of infective conjunctivitis may be divided into two broad clinical groups : acute, and subacute or chronic. Acute conjunctivitis may be classified as serous, catarrhal, muco-purulent, purulent and membranous. Subacute or chronic conjunctivitis includes simple chronic conjunctivitis, angular conjunctivitis and follicular conjunctivitis, while specific clinical pictures are associated with trachoma, tuberculosis, syphilis and tularemia.

It is interesting that in cases of conjunctivitis which appear to be of infective origin, the majority shows no significant organismal contaminants. At the time of writing, in London, bacteriological examination shows that some 25 per cent. of cases are clinically infected, 10 per cent. being due to staphylococci, 7 per cent. to pneumococci, and 3 per cent. to *H. ægyptius*; 75 per cent. are bacteriologically sterile. On the average, 35 per cent. of the latter show the presence of viruses, of which 17 per cent. are herpes and 10 per cent. various types of adenoviruses.

Serous Conjunctivitis is characterized by a minimal degree of congestion, a watery discharge, and a diffuse boggy swelling of the mucous membrane. It is typically caused by a mild viral infection such as does not give rise to a follicular response (adenovirus, etc.).

Acute Catarrhal or Muco-purulent Conjunctivitis. In its milder forms an infection of the conjunctiva assumes the characteristics of a typical catarrhal inflammation of a mucous membrane. The picture of hyperæmia is associated with a mucous discharge which gums the lids together, particularly in the mornings because of the accumulation during the night. The condition is commonly but erroneously attributed to a "cold in the eye".

In the more severe cases the whole conjunctiva is a fiery red ("pink eye"); all the conjunctival vessels are congested, a phenomenon less marked in the circumcorneal zone (Plate V, Fig. 1; Fig. 135). Flakes of muco-pus and eventually of pus are seen in the fornices and often on the margins of the lids, matting the lashes together with dirty yellow crusts. This appearance may be easily mistaken for blepharitis, but if the crusts are bathed off, the underlying lid margins will be found to be healthy. Flakes of mucus passing across the cornea may give rise to coloured halos, owing to their prismatic action. These "halos" must be carefully distinguished from those met with in glaucoma (*q.v.*).

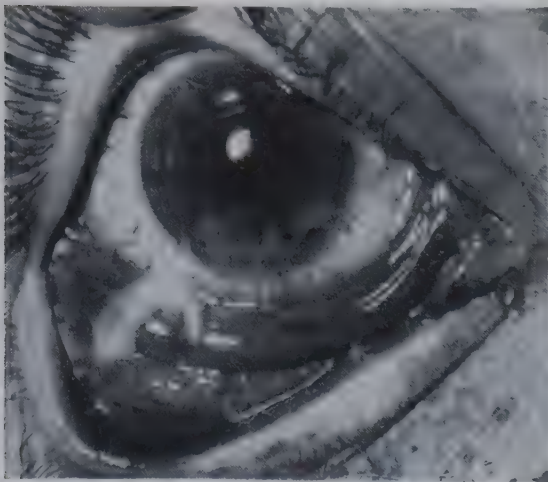


FIG. 135. Muco-purulent conjunctivitis.

The disease reaches its height in three or four days : if untreated it is liable to pass into a less intense, chronic condition. Complications are rare, but abrasions of the cornea are liable to become infected and to give rise to ulcers. Occasionally marginal ulcers form or a superficial keratitis may develop.

Ætiology. Muco-purulent conjunctivitis is caused by a number of organisms and is contagious, being transmitted directly by the discharge.

Among the most common ætiological organisms is the staphylococcus which may also be responsible for other associated conditions such as blepharitis and eczema or impetigo of the skin ; it is frequently associated with multiple corneal erosions or other forms of superficial punctate keratitis. The usual source of organisms is the nose. Many other organisms may be responsible for the disease but two deserve special mention.

The *H. ægyptius* of Koch-Weeks is a very slender rod, varying much in length, Gram-negative, staining badly with the ordinary basic dyes. Groups of bacilli found in much degenerated "skeletonized" pus cells are characteristic. The organism may give rise to widespread epidemics particularly in sandy, semi-tropical countries often associated with severe corneal involvement, but in temperate countries the cases are usually mild and sporadic, although contact transmission is common. An attack confers immunity for some time.

Pneumococcal conjunctivitis is not definitely separable from the other acute forms clinically, but there is usually more œdema (chemosis), small ecchymoses are common, and a membranous film may form—"pseudo-membranous conjunctivitis." It ends in a crisis, like pneumococcal infection of the lungs, after which the organism rapidly disappears from the secretion, but may be accompanied by nasal catarrh, which may precede or follow the inflammation. Iritis is rare as a sequel of conjunctivitis, but pneumococcal conjunctivitis is exceptional in this respect and if the cornea is involved a hypopyon ulcer may develop.

Muco-purulent conjunctivitis generally accompanies exanthemata such as measles and scarlet fever.

Treatment. The treatment of muco-purulent conjunctivitis consists of two main procedures : (a) the frequent washing out of the conjunctival sac with a suitable lotion ; and (b) the control of the infection by appropriate drugs. The eyes should not be bandaged, as this prevents the free exit of the secretion, but if there is any photophobia a shade or dark goggles should be worn.

In irrigating the conjunctival sac eye lotions act chiefly by washing out deleterious material, since they cannot be used in sufficiently strong concentrations to act as efficient antiseptics. Although some effect may be gained by using solutions such as hydrarg. oxycyanide (App. I), on the whole, normal saline solution can as well be ordered. It is useless to bathe the eyes unless the fluid irrigates the conjunctival sac. In every case the lotion should be warmed by the addition of hot water.

The patient may be directed to use the ordinary eye-bath for the application of eye lotions. If a child, the parents should be instructed to hold the lids apart, the child lying upon its back ; a pad of cotton-wool, dripping with the lotion, which should be warm, is then held over the eye and the lotion squeezed out : the process is repeated until all discharge has been washed away. For thorough irrigation of the conjunctival sac, however, an "undine" (Fig. 136) is the most effective reservoir. The lids are everted and the lotion

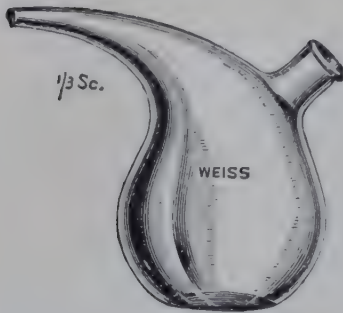


FIG. 136. Undine.

is poured from a little height over the whole surface, every crevice being irrigated as thoroughly as possible. The nozzle of the undine should not be allowed to touch any part of the eye.

Control of infection is most effectively maintained by the use of bacteriostatic drugs. Ideally the appropriate drug should be chosen after tests of bacterial sensitivity have been made. In default of this, one or other of the "broad-spectrum" antibiotics is the most generally useful (p. 143). The value of these drugs is greatest in the acute stage ; when the infection has become chronic their effect is frequently temporary and relapses are common. In these circumstances they cannot entirely replace the older methods of treatment. An antibiotic ointment or soft yellow paraffin is smeared along the lids at bed-time or, in the case of children, as often as they are put to sleep : it prevents the lids from sticking together—a two-fold benefit, that of preventing discharge from being retained, and that of obviating pain on opening them.

For the sake of cleanliness ointments should be prescribed in collapsible tubes from which they are readily applied by expressing a small quantity directly into the lower fornix. If dispensing in tubes is not available, a glass rod is the best vehicle (Fig. 137) ; the surgeon should first pass it through his fingers lest it be chipped. The patient, if a child, is placed upon his back



FIG. 137. Glass rod.

on a couch and an assistant holds the arms against the body keeping the legs still by pressure with the elbows. The surgeon separates the lids with two fingers of one hand and places the end of the glass rod carrying the ointment between the separated lids. Keeping the rod in position, the lids are allowed to close upon its end, and it is then withdrawn by carrying it outwards towards the temple. The other end of the rod and the surgeon's other hand are used for the other eye.

If this treatment is properly carried out, the patient will usually be well in a few days. If, however, the case is not progressing satisfactorily and if sensitivity tests do not give a definite lead for a change in antibiotic drug, the old-fashioned but nevertheless efficacious treatment of painting the lids once with silver nitrate

may be tried. This technique may also be useful in localities where the full facilities of bacteriology and modern medicine are not available ; moreover, it is sometimes efficacious at a stage when an acute inflammation shows signs of becoming chronic.

Silver nitrate should be used in a solution of strength of 1 per cent. since this is the weakest dilution at which its coagulant action is effective. It acts by forming a thin epithelial eschar and coagulating the muco-purulent discharge which is cast off in flakes. The irritation is reduced if the lid is kept everted for a few minutes and the flakes gently removed with cotton-wool, a drop of 2 per cent. cocaine solution being then instilled. Silver nitrate is not strongly bactericidal, but many organisms are entangled in the coagulum and removed with it. Moreover, there is a powerful physiological response to the caustic ; hyperæmia increases and the tissues are flooded with blood serum, which can thus more effectively exert its bactericidal powers.

The best method of painting the lids is with the use of a glass rod (Fig. 137). A *very thin* wisp of cotton-wool is then tightly wound round the end after it has been made damp. The end of the wisp is left loose, so that it may absorb the solution. The lids are everted ; the wool, dipped in the silver solution, is applied freely to the conjunctival sac, the cornea being protected as much as possible. It is unnecessary to neutralize the excess of nitrate with salt solution, as is often taught. The excess may be mopped up with a pad of dry absorbent wool. If, as is usually the case, the other eye is affected, the other end of the rod is used in the same manner. In the absence of a glass rod the best implement is an ordinary wooden match, used in the same way.

After an attack of muco-purulent conjunctivitis the conjunctiva generally returns to a normal condition. If the case has been neglected and chronic inflammatory signs persist, treatment should be as for chronic conjunctivitis (*q.v.*).

Since the disease is contagious care must be taken to prevent its spread. The patient must keep his hands clean and no one else must be allowed to use his towel, handkerchief, or other fomites.

Purulent Conjunctivitis (*Acute Blennorrhæa*) is a much more serious condition. It occurs in two forms—as ophthalmia neonatorum in babies, and as conjunctivitis in the adult. Many cases are caused by the gonococcus but the same clinical picture may be found with staphylococci, streptococci, *C. diphtheriæ*, and with mixed infections.

GONORRHOËAL CONJUNCTIVITIS. Fortunately, in medically advanced countries gonorrhœal conjunctivitis is now comparatively rare.

The *Neisseria gonorrhœæ* is a bun-shaped diplococcus, staining readily, decolorized by Gram and found within both leucocytes and epithelial cells. The *N. catarrhalis* and the *N. meningitidis*, both Gram-negative, are sometimes found in the conjunctival sac. They may be distinguished from the gonococcus by agglutination tests. The *N. catarrhalis* is rarely found in acute conjunctivitis, but more often in chronic and post-operative forms.

The disease is acute and in adults is due to direct infection from the genitals, occurring usually in males and first in the right eye. There is much swelling of the lids and conjunctiva, a copious purulent

discharge, a marked tendency to involvement of the cornea, and constitutional disturbances including a rise of temperature and mental depression (Plate V, Fig. 3).

The incubation period is a few hours to three days. Thereafter the upper lid becomes swollen and tense, overhanging the lower, and edged with pus. Eversion, which is difficult, shows that the palpebral conjunctiva is deep red and velvety; rarely there is a membrane. There is great pain and the pre-auricular lymph node is enlarged and tender and may suppurate. After two or three weeks the purulent discharge diminishes, but subacute conjunctivitis with much papillary thickening of the conjunctiva persists for several weeks longer. The gonococcus is still present—a point of great importance, both as regards contagion and treatment. No immunity is conferred by the attack.

The most important point in diagnosis is the coincidence of urethritis. The most important point in prognosis is the condition of the other eye.

Corneal complications are the rule, and constitute the causes of blindness. There may be diffuse haziness of the whole cornea, with grey or yellow spots near the centre. Ulcers may occur at any part, and are due to necrosis of the epithelium through direct invasion by the organisms. Marginal ulceration, which may extend completely round the cornea, may be due to retention of pus in the angle formed by the chemotic conjunctiva. When ulceration has commenced it progresses rapidly and deeply and perforation is common with all its attendant dangers. Ulceration commencing late is not so dangerous. The greatest care should therefore be taken to prevent injury to the cornea during the manipulation necessary for diagnosis and treatment; abrasions which may ulcerate are easily produced by the finger nails and even by the rough use of wool swabs.

Iritis and iridocyclitis, with attendant complications, may arise independently of perforation of the cornea, and lead to serious diminution of vision. Gonorrhœal arthritis is not uncommon, and endocarditis and septicæmia may arise as complications.

Treatment should be directed first to protection of the other eye. Several drops of a solution of penicillin or other suitable antibiotic are instilled every few minutes for some hours; if these are not immediately available this eye should be given a protective covering.

If pus from a gonorrhœal conjunctivitis spurts accidentally into the surgeon's eye, the conjunctival sac should be freely irrigated and the same antibiotic treatment should be initiated. To prevent such an accident every attendant on a gonorrhœal case should wear protective goggles.

If the disease is established and there is any purulent discharge, the eye must be irrigated with warm saline and intensive therapy with penicillin (or another suitable antibiotic) started, using drops

ACUTE CONJUNCTIVITIS



FIG. 1. Mucopurulent conjunctivitis. FIG. 2. Angular conjunctivitis.



FIG. 3. Purulent (gonococcal) conjunctivitis.



FIG. 4. Pseudo-membranous (streptococcal) conjunctivitis.



FIG. 5. Membranous (diphtheritic) conjunctivitis.

[To face p. 156.]

in a concentration of 5,000 units per ml. every minute, for half an hour. Repeated irrigations are unnecessary since, in the first place, penicillin remains effective in the presence of pus and, in the second, the discharge rapidly disappears. Any pus that does accumulate is wiped away with moist pledgets of cotton-wool. Penicillin drops are continued at five-minute intervals for a further half-hour, and the treatment consolidated by half-hourly and then hourly instillations for two days or so. Astringent lotions are then employed (App. I).

An alternative but less efficacious, although a useful supplementary method of treatment, is the systemic administration of a full course of such sulphonamides as sulphadiazine.

In default of treatment by antibiotics, or when dealing with insensitive organisms, reliance must perforce be placed on the old-fashioned treatment of repeated applications of silver nitrate together with repeated irrigations (2-hourly during the day and 4-hourly during the night) with antiseptic lotions. Such treatment, however, is most unsatisfactory; corneal complications frequently supervene and recovery is often slow.

Atropine should be used in all cases in which the cornea is involved since this is always accompanied by some iritis; corneal complications require very active treatment (*q.v.*).

METASTATIC GONORRHOEAL CONJUNCTIVITIS sometimes occurs as an endogenous infection in adults, associated with gonorrhœal arthritis. It is a mild simple conjunctivitis, generally bilateral, and occasionally accompanied by iritis. It usually responds readily to local treatment, but is apt to recur if the arthritis relapses.

Ophthalmia neonatorum is a preventable disease occurring in new-born children due to maternal infection acquired as the result of carelessness at the time of birth; it used to be responsible for 50 per cent. of blindness in children, but recently the decline in the incidence of gonorrhœa as well as effective methods of prophylaxis and treatment have almost eliminated its occurrence and the seriousness of the sequelæ in advanced communities. The virtual elimination of this disease has constituted a revolution in ophthalmology within this generation and today the disease is usually mild, due to the *Chlamydia oculogenitalis*, staphylococci or other organisms. Any discharge, even a watery secretion, from a baby's eyes during the first week should be viewed with suspicion, since tears are not secreted at this early date.

In cases of virulent *gonococcal* infection the discharge rapidly becomes muco-purulent and then purulent. Both eyes are nearly always affected, though one is usually worse than the other. The conjunctiva becomes intensely inflamed, bright red, and swollen, and pours out thick yellow pus. Marked chemosis is a distinguishing feature from severe muco-purulent conjunctivitis, and when the lids

are separated by retractors the cornea is seen at the bottom of a crater-like pit. There is dense infiltration of the bulbar conjunctiva, and the lids are swollen and tense. Later the lids become softer and more easily everted, the conjunctiva becomes puckered and velvety, and the blood stasis gives place to intense congestion, with the free discharge of pus, serum and often blood. In some cases a false membrane forms, so that the case resembles a membranous conjunctivitis.

There is great risk of corneal ulceration in untreated gonococcal ophthalmia neonatorum, since this organism has the power of invading intact epithelium. The slightest haziness of the cornea should be viewed with apprehension. Sometimes the cornea is already ulcerated, and not infrequently perforated, when the child comes under observation. Ulceration usually occurs over an oval area just below the centre of the cornea, corresponding to the position of the lid margins when the eyes are closed and consequently rotated somewhat upwards. More rarely oval marginal ulcers are formed as in the gonorrhœal conjunctivitis of adults. The ulcers extend rapidly, both superficially and in depth, and perforation occurs, usually indicated by a black spot or area in the ulcer, caused by a prolapse of the iris. Sometimes perforation is sudden, a large part of the iris prolapses, and the lens may be extruded, while in the worst cases there is a black hole in the cornea filled with clear vitreous.

Metastatic stomatitis and arthritis rarely occur. The arthritic manifestations usually appear in the third or fourth week and affect the knee, wrist, ankle or sometimes elbow. The course is benign, abscesses being rare.

The baby's eyes must be examined as described on p. 94, retractors being used to separate the lids and the surgeon wearing protective goggles lest pus spurt into his eyes when the child's lids are separated. A bacteriological examination should be made in every case.

In inadequately treated cases serious sequelæ may occur. If the corneal ulceration heals without perforation there is always much scarring of this tissue, but the nebula clears more in babies than in older people. Perforation may be followed by anterior synechiæ, adherent leucoma, partial or total anterior staphyloma, anterior capsular cataract or panophthalmitis. When vision is not completely destroyed but is seriously impaired by the corneal opacities, the development of macular fixation which takes place during the first six weeks of life is impaired, resulting in the development of nystagmus which persists throughout life; this may not become manifest until a later date.

Inclusion conjunctivitis is a relatively common cause of ophthalmia neonatorum in Western countries. Bacterial examination is negative or inconsequential but the characteristic intracellular inclusion

bodies formed by the *Chlamydia oculogenitalis* (a Bedsonian organism) are found (Fig. 138). It is a venereal infection derived from the cervix or urethra of the mother. The inflammation is much less severe than the gonococcal type but the conjunctiva may be considerably swollen and œdematous while the discharge may be purulent. In the absence of a subconjunctival adenoid layer at this age there are, however, no follicles as appear in this infection in the adult, but if the disease is allowed to smoulder into a chronic stage these may develop after three months. A complicating superficial keratitis is the rule and occasionally in prolonged cases the corneal periphery may be invaded by pannus.

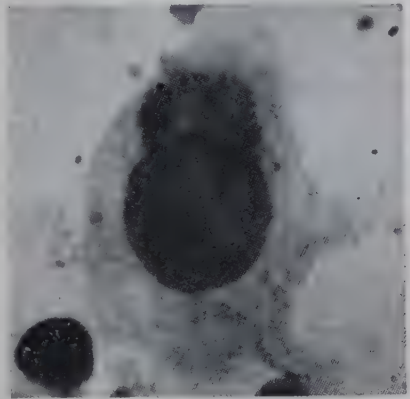


FIG. 138. Epithelial cell showing inclusion body lying above the nucleus. Giemsa staining.

Treatment. The disease is preventable ; *prophylactic* treatment is therefore of prime importance. Any suspicious vaginal discharge during the antenatal period should be treated, and the most meticulous obstetric asepsis maintained at birth. The new-born baby's closed lids should be thoroughly cleansed and dried. If infection is suspected a drop of silver nitrate solution, 1 per cent., may be instilled into each eye (Credé's method). The eyes must be carefully watched during the first week.

Some considerable controversy still exists over this method of prophylaxis. Many authorities advocate the use of penicillin or other antibiotic drugs but all the organisms which may be present may not be sensitive to the drug employed. If silver nitrate is used the solution should never be stronger than 1 per cent. lest corneal opacities result.

If the disease is established the treatment should be on the lines already indicated for gonorrhœal ophthalmia ; fortunately most of the types of infection in ophthalmia neonatorum, including the Bedsonian infection, are amenable to penicillin. The eyes are washed out with saline and if the cornea is involved atropine is instilled. Thereafter intensive treatment with penicillin (or other antibiotic) is adopted as already described. After about half an hour the clinical picture has altered and although there is some swelling

of the lids and conjunctiva, the eye is dry. After a further twenty-four hours' treatment a clinical cure is usually obtained.

Before the advent of penicillin, general sulphonamide therapy was the treatment of choice (p. 141) : it is most efficient both for the organismal and Bedsonian infections and should always be adopted in those rare instances in which the organism is insensitive to penicillin. Sulphadimidine and sulphadiazine are well tolerated by infants. It is to be noted that sulphonamides used locally are ineffective. These drugs are a great advance over the classical methods of treatment by repeated irrigation and painting with silver nitrate. While these latter methods took weeks to check the infection, the sulphonamides take as many days: penicillin, on the other hand, *when used intensively*, brings about a cure in as many hours.

Membranous Conjunctivitis (*Diphtheritic Conjunctivitis*). As in inflammation of the throat, the conjunctival surface may become covered by a fibrinous membrane ; and similarly, the milder croupous clinical varieties in both can be distinguished from the more severe diphtheritic. It has been placed beyond dispute, however, that mild cases may be diphtheritic, and severe non-diphtheritic ; hence it is best to speak simply of membranous conjunctivitis until a bacteriological examination has been done. A variety of organisms other than the diphtheria bacillus, such as the pneumococcus or streptococcus, can produce a membrane, especially in weakly children, particularly after measles and scarlet fever and in association with impetigo ; these cases are sometimes called pseudo-membranous but they cannot be distinguished clinically with certainty. The condition occurs chiefly in children who have not been immunized, and shows all degrees of severity.

In mild cases there is some swelling of the lids and a mucopurulent or sanious discharge. On everting the lids the palpebral conjunctiva is seen to be covered with a white membrane which peels off rapidly without much bleeding (Plate V, Fig. 5).

In severe cases the lids are more brawny ; the conjunctiva is permeated with semi-solid exudates, which impair mobility, compress the vessels, prevent the formation of a free discharge, and tend to necrosis both of the conjunctiva and cornea. In these cases the membrane separates less readily, the underlying surface bleeding unless it is too infiltrated and solid. The membrane may be patchy or cover the whole palpebral conjunctiva, often beginning at the edge of the lid, but is seldom found on the bulbar conjunctiva. The pre-auricular lymph node may be enlarged and may suppurate. The temperature is raised unless the patient is in a moribund condition, and albumin is frequently present in the urine.

For six to ten days there is great peril to the cornea from ulceration usually due to secondary infection. About the same time, also, the sloughs begin to separate and the discharge becomes more

profuse. In a few days the conjunctiva assumes a red and succulent appearance and there is danger of adhesions forming between the palpebral and bulbar parts of the conjunctiva (symblepharon).

Post-diphtheritic paralyses, even of accommodation, are rare.

Cases of less severe but more chronic *pseudo-membranous* conjunctivitis are occasionally met with (Plate V, Fig. 4). In *ligneous conjunctivitis* the membrane is cast off, but recurs again and again; the pathology of these cases is not understood. Membrane formation may also occur as a complication of erythema multiforme.

Pathology. There is little or no relationship between the severity of the local condition and the presence or absence of the *C. diphtheriæ*. Owing to the difficulty of distinguishing this organism from the *C. xerosis*, with which it is morphologically identical, inoculation tests are the only absolutely reliable diagnostic criterion. It is rare to obtain evidence of primary diphtheria of the throat. Other bacteria which occasionally form membranes are the pneumococcus, streptococcus, *H. ægyptius*, gonococcus, staphylococcus, *E. coli*, etc. Streptococcal conjunctivitis, a very virulent form, occurs chiefly in children, associated with measles, scarlet fever, whooping cough, and influenza.

Other cases may be due to the action of heat, caustics, and other non-bacterial causes.

Treatment. Every case should be treated as diphtherial unless good negative evidence is afforded by films and cultures. This consists of the intensive local and general administration of penicillin together with the prompt injection of anti-diphtheritic serum (4-6-10,000 units repeated in twelve hours). Antitoxin given early and in adequate amounts both locally and systemically is curative, but it should be combined with the antibiotic.

In *streptococcal membranous conjunctivitis* the danger of necrosis of the cornea and even of the death of the patient is considerable, so that immediate local and general treatment is necessary. Depending on the sensitivity of the organism this should be carried out with the intensive topical and systemic use of a suitable antibiotic (Chap. 14) or, alternatively, the local use of the antibiotic may be supplemented by a full course of treatment by one of the less toxic sulphonamides.

Simple Chronic Conjunctivitis occurs as a continuation of simple acute conjunctivitis, sometimes in spite of orthodox treatment. It is frequent when a cause of irritation is continuous—smoke, dust, heat, bad air, late hours, abuse of alcohol, and so on—or when it is caused by hypersensitivity to an allergen which has not been eliminated. Permanent irritation from concretions (p. 178) in the palpebral conjunctiva, misplaced lashes, dacryocystitis and chronic rhinitis must be remembered and as far as possible eliminated. Unilateral chronic conjunctivitis should suggest the presence of a foreign body retained in the fornix, or inflammation of the

lacrimal sac. It is often necessary to make a thorough and systematic investigation of the local and general condition before the cause can be found. It is not infrequently associated with chronic intranasal trouble, and seborrhœa, particularly of the scalp; dandruff is a common accompaniment. The disease is too frequently regarded as trivial, but may be a source of great discomfort.

The essential symptoms are burning and grittiness, especially in the evening when the eyes often become red while the edges of the lids feel hot and dry. Difficulty in keeping the eyes open is a common symptom. The lids may or may not be stuck together on waking for the discharge is slight, but there is frequently an abnormal amount of secretion from the meibomian glands.

Superficially the eyes may look normal but when the lower lid is pulled down the posterior conjunctival vessels are seen to be congested, and the surface of the mucous membrane is sticky. The palpebral conjunctiva, upper and lower, may be congested, with a velvety papilliform roughness; this is due to a hypertrophy of the normal vascularized papillæ in the submucosa. Occasionally it is succulent and fleshy.

Treatment consists in eliminating the cause and restoring the conjunctiva to its normal condition. Errors of refraction and chronic nasal catarrh are perhaps most likely to be forgotten; they should be sought out as a matter of routine. When heat is a prominent ætiological factor (as in cooks or in industry) protective glasses may be ordered. The treatment of the special local conditions mentioned above will be discussed in their proper place. A swab should be taken to eliminate the presence of infective organisms and it is well at the same time to determine the bacteriological flora of the nose and upper respiratory passages; any conjunctival or nasal infection should receive the proper treatment. It is to be remembered that a chronic infection of the upper respiratory tract will frequently make a chronic conjunctivitis persist indefinitely unless remedial measures are directed to the primary seat of infection; in this connection the effect of the toxoid in a staphylococcal infection should be kept in mind. In such cases vaccine therapy may be indicated.

Local treatment consists firstly in eliminating any infection by a *short* course of a suitable antibiotic and then in diminishing congestion and restoring the conjunctiva to its normal suppleness and secretory activity. The condition is largely a lack of tone, due to defective response to prolonged irritation; a stimulating treatment is therefore indicated as supplied by astringent applications. In mild cases weak astringent lotions suffice (App. I); they should be used two or three times a day, not immediately before going to bed. Adrenaline has a transient effect in diminishing redness and itching. Boric ointment or soft yellow paraffin should be applied to the margins of the lids at bed-time. In recalcitrant cases mercury oxycyanide (1 in 10,000) may be used, followed by zinc sulphate

lotion at a later stage. In more severe cases a preliminary painting with silver nitrate solution may be of value and may be repeated once or twice, but silver preparations should not be ordered for application at home, since prolonged use may lead to staining of the conjunctiva (argyrosis). When there is an abnormal amount of secretion from the tarsal glands (*conjunctivitis meibomiana*), this should be squeezed out of the glands by repeated massage of the lid with the thumb against a spatula laid upon the conjunctival surface.

Angular Conjunctivitis (*Diplobacillary Conjunctivitis*). In this condition the reddening of the conjunctiva is limited almost exclusively to the inter-marginal strip, especially at the inner and outer canthi, and to the bulbar conjunctiva in the same neighbourhood (Plate V, Fig. 2); there is also excoriation of the skin at the inner and outer palpebral angles, which may be very slight—a mere scurfiness—but is nearly always present. There is discomfort, with slight muco-purulent discharge and frequent blinking. If untreated the condition becomes chronic and may give rise to blepharitis. Clear, shallow, corneal ulcers may occur, but are rare; they are usually marginal, but may be central and associated with hypopyon (*q.v.*). A single attack does not confer immunity, and relapses are not uncommon.

Pathology. Such a condition may be caused by the staphylococcus but is typically due to the *Moraxella*, a diplobacillus consisting of pairs of large, thick rods, placed end to end which stain well with basic stains, are decolorized by Gram, and are easily recognized in films. They produce a proteolytic ferment which acts by macerating the epithelium. There is an incubation period of four days. The diplobacilli are strongly resistant to drying. They have been found in the nasal tract of healthy persons, and are often present in the nasal discharge in cases of angular conjunctivitis.

Treatment. Diplobacillary conjunctivitis responds to oxytetracycline applied as ointment (p. 561). Although less rapidly effective a zinc lotion (p. 560) is of great value; this acts by inhibiting the proteolytic ferment. Zinc oxide ointment may be applied to the lids at night.

Follicular Conjunctivitis. The occurrence of follicles in the conjunctiva either as an acute, subacute or chronic manifestation of disease is relatively common; they do not occur in the normal conjunctiva. Such follicles appear as rounded swellings, 1 to 2 mm. in diameter, and are due to localized aggregations of lymphocytes in the subepithelial adenoid layer, and unless an acute inflammation is present, the conjunctiva over them remains normal (Fig. 139). In all types of follicular conjunctivitis the histological nature of the follicles is identical, but in trachoma degenerative changes and eventually scarring distinguish the condition from non-trachomatous types.

It is important to differentiate follicles from papillæ—a hyperplasia of the normal system of vascularization with glomerulus-like bunches of capillaries of new formation growing into the epithelium in inflammatory conditions. Both give rise to a roughened appearance of the conjunctiva and the slit-lamp may be necessary for their clinical differentiation. It is interesting that the conjunctiva of the newborn is unable to produce follicles before 2 or 3 months of age, so that an infection very early in life may appear initially as a papillary conjunctivitis and may develop into follicular conjunctivitis if it remains active for longer than 3 months.

Follicular hypertrophy may be due to certain chemicals and toxins, the most notable of which is eserine after prolonged use ; a milder manifestation may follow the use of pilocarpine and other drugs such as DFP. The ætiology of several types of follicular con-

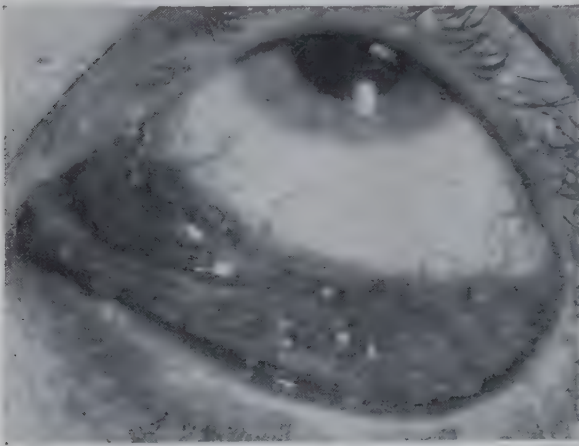


FIG. 139. Follicular conjunctivitis.

junctivitis is obscure, but this conjunctival reaction is most commonly caused by viruses, particularly those of herpes and the adenoviruses. Isolated follicles, however, may occur particularly in the lower conjunctiva in any conjunctivitis of long standing.

FOLLICULOSIS is a common condition occurring particularly in young children. A follicular hypertrophy occurs bilaterally, involving primarily the conjunctiva of the lower lid ; here follicles, often of considerable size, are arranged in parallel rows, but they are rarely seen in the upper fornix and never on the bulbar conjunctiva. Between the follicles the conjunctiva retains its normal transparency and apart from a slight irritation and occasionally increased sensitivity to light, the condition is symptomless and secretion is absent. They may persist for several years uninfluenced by treatment but resolution without sequelæ is invariable. Folliculosis occurs usually in young people who are not robust and show an accompanying hypertrophy of the lymphoid tissue of the upper respiratory passages ; indeed, it is generally considered to be a manifestation of the same tendency which produces adenoid vege-

tations ; but the isolation of adenoviruses from the throat of some cases may perhaps have some ætiological significance.

Treatment consists of the use of weak astringent lotions, while the general health should receive attention.

ACUTE FOLLICULAR CONJUNCTIVITIS. Several types of acute follicular conjunctivitis occur.

Inclusion conjunctivitis is characterized by a relatively acute onset, the incubation period varying from 5 to 10 days. The follicular hypertrophy is always more prominent in the lower lid than the upper, although in acute cases the follicles may be partially obscured by papillary hypertrophy. The exudate, composed principally of neutrophils, may be moderately abundant, and the cornea is involved in a superficial punctate keratitis occasionally with some pannus-like peripheral vascularization.

The disease is caused by an organism resembling that of trachoma and producing inclusion bodies morphologically identical with those occurring in this disease (Fig. 138). The primary source of infection is a benign subclinical venereal disease producing a mild urethritis in the male and a cervicitis in the female ; it is commonly transmitted to the newborn from the mother (p. 157). In adults the organism may be transferred from the genitals by the fingers, but a common mode of infection is through the water of swimming pools so that the disease may occur in local epidemics (*swimming-bath conjunctivitis*).

The disease runs a relatively benign course, healing spontaneously when untreated in from 3 to 12 months ; the organism, however, is very responsive to treatment with the broad-spectrum antibiotics or systemic treatment by the sulphonamides.

Epidemic kerato-conjunctivitis. This disease is characterized by a rapidly developing follicular conjunctivitis with marked inflammatory symptoms and scanty exudate, associated with a pre-auricular adenopathy. Occasionally it takes on a membranous form. Seven to ten days after the infection, corneal complications appear—initially punctate epithelial infiltrates followed by the development of discrete subepithelial opacities associated with photophobia. The conjunctival manifestations gradually diminish and finally disappear but the corneal opacities may persist for many months or even years. The condition is markedly contagious and occurs in widespread epidemics, unfortunately often disseminated in clinics by contaminated solutions, fingers or tonometers. It has been associated with several types (3, 7 and particularly 8) of the adenoviruses. Treatment by antibiotics or otherwise is relatively ineffective so that it may entail prolonged disability.

Pharyngo-conjunctival fever is characterized by an acute follicular conjunctivitis in association with pharyngitis and fever and occasionally with pre-auricular adenopathy, appearing chiefly in children in epidemic form. Corneal involvement as a superficial punctate keratitis is rare.

The disease is acute and transient and antibiotics have little effect. It is caused by one or other of the group of adenoviruses. It is probable that it represents the acute conjunctivitis formerly known as *Béal's conjunctivitis* which runs a similar short self-limiting course.

Newcastle conjunctivitis is clinically indistinguishable from the conjunctivitis of pharyngo-conjunctival fever ; it is caused by the Newcastle virus derived from contact with diseased fowls.

Acute herpetic conjunctivitis occurs as a primary manifestation of herpes ; it is thus usually seen in young children who are as a rule infected by contagion from carriers of the virus. It is comparable with the more common acute stomatitis which results from an initial herpetic infection and may be associated with the usual vesicular lesions on the face. A pre-auricular adenopathy is present and the corneal vesicles which may merge to form dendritic figures (*q.v.*) are frequent. The condition is acute, the follicles are usually large, and the corneal scarring tends to be permanent.

Trachoma, once known as Egyptian ophthalmia and endemic in the Middle East since pre-historic times, was spread far and wide in Europe by the French armies during the Napoleonic wars. It is now endemic in many parts of the world, particularly Eastern and Central Europe, the Middle East, Central and Eastern Asia (Persia, India, China and Japan), Indonesia, the Pacific Islands, North and Central Africa, Central, and large areas of South America. It shows a predilection for certain races such as Jews, Australian aborigines and North American Indians, but it is not a racial disease and no race is exempt ; in Western countries it is rare. It has been estimated that about one fifth of the inhabitants of the world are affected, and trachoma together with the complicating infections with which it is associated is the cause of more blindness than any other condition.

The disease flourishes among people whose surroundings are unhygienic and who are crowded together in an unhealthy environment wherein dirt abounds. In endemic areas children are infected often in the first few years of life. It is contagious in its acute stages, being spread by the transference of conjunctival secretion by such means as fingers or towels and, above all, by flies, a liability increased by the presence of much discharge. On the other hand, scrupulous cleanliness will prevent extension of the disease to healthy subjects.

The disease usually starts subacutely, but on massive infection, experimental, accidental or clinical, an acute onset may be observed. Its course is determined largely by the presence or absence of a complicating infection. In the absence of such an infection a "pure" trachoma may be a relatively mild disease, so mild and symptomless, indeed, as to excite little or no attention until perhaps cicatrization manifests itself in later life ; in such cases the discovery of follicles or other cicatricial remnants on the upper tarsal conjunctiva when the lid is everted may come as a surprise to the patient and his relatives. On the other hand, in many countries

wherein the disease is endemic, particularly in North Africa and the Middle East, secondary infections (as by *H. ægyptius*, the gonococcus or other organisms) result in an acute and incapacitating condition liable to relapses and leading to gross cicatricial sequelæ which often end in blindness. In those countries where an acute conjunctivitis frequently occurs in epidemic form, the clinical onset of trachoma first appears after the acute conjunctivitis has subsided. In many cases, therefore, the so-called "acute" trachoma is a subacute infection upon which an acute muco-purulent or purulent conjunctivitis has been superimposed.

The primary infection is epithelial and involves both the conjunctiva and the cornea. The typical *conjunctival signs* are the appearance of a diffuse inflammation characterized by congestion, papillary enlargement and the development of follicles. The conjunctiva covering the upper tarsus is usually most affected and appears red and velvety, a condition which may pass

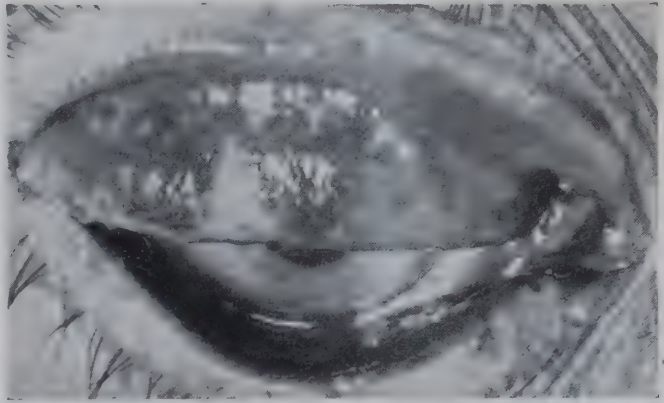


FIG. 140. Trachoma:
follicular stage.

into a uniform jelly-like thickening. The essential lesion is the trachoma follicle. When small, these cannot be distinguished from those of follicular conjunctivitis but they often assume a size (up to 5 mm. in diameter) and appearance never seen in non-trachomatous conditions. Their distribution is characteristic. They may commence in the lower fornix but in most cases they quickly appear in the upper fornix also where they are usually most accentuated, often forming a row along the upper margin of the tarsus (Fig. 140). They are not, however, limited to these regions but may appear on the caruncle and the plica as well as over the palpebral conjunctiva generally. They are rare on the bulbar conjunctiva but when seen here are pathognomonic of trachoma. Invasion of the lacrimal passages is not common. Trachomatous infiltration may spread deeply into the subepithelial tissues of the palpebral conjunctiva and even invade the tarsal plate. An important diagnostic feature is the appearance at a relatively early stage of signs of cicatrization of the follicles, often appearing as minute star-shaped scars visible with the slit-lamp.

Trachomatous *implication of the cornea* manifests itself initially as a superficial keratitis, usually of so slight a degree as to be evident only by slit-lamp examination after staining with fluorescein. It occurs typically in the upper part of the cornea where there are numerous epithelial erosions which later become associated with infiltrated areas in the substantia propria.

At a later stage *trachomatous pannus* develops as a lymphoid infiltration with vascularization of the margin of the cornea, usually limited to the upper half (Fig. 141) but tending to spread towards the centre and to involve the whole cornea. The upper part of the margin of the cornea becomes cloudy, and minute superficial vessels, springing from the corneal loops, grow inwards towards the centre. The haziness and vascularization increase until the upper half of the cornea is affected. At the same time follicle-like infiltrations may appear near the limbus (Herbert's pits). The vessels are all superficial (p. 103), and microscopic examination has shown that

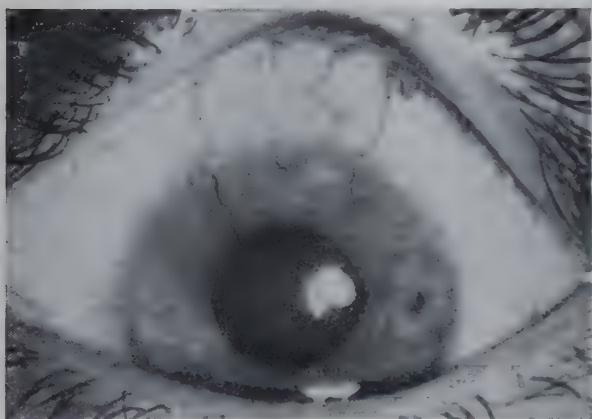


FIG. 141. Trachomatous pannus.

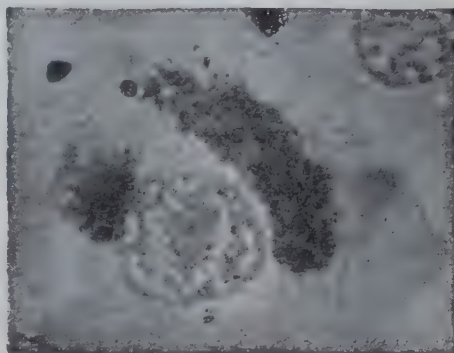
they lie at first between Bowman's membrane and the epithelium, carrying in with them a small amount of granulation tissue. In the later stages Bowman's membrane disappears and the superficial layers of the substantia propria become involved. In more severe cases the vascularization is not limited to the upper part, but superficial vessels grow in from all sides and the whole cornea becomes vascularized and opaque.

In progressive pannus the vessels are mostly parallel to each other and directed vertically downwards, anastomosing little. They extend to a level which forms a horizontal line, and beyond this line there is a narrow strip of infiltration and haze. In regressive pannus, on the other hand, the vessels extend a short distance beyond the area which is infiltrated and hazy: this difference is useful in estimating the results of treatment. *Corneal ulcers* which may be chronic and indolent may occur anywhere but are commonest at the advancing edge of the pannus. They are shallow, little infiltrated and very irritable, causing much lachrimation and photophobia.

Pannus may resolve completely, leaving the cornea quite clear apart from fine obliterated vessels, but only in cases treated early when the pannus has not destroyed Bowman's membrane. In other cases a permanent opacity results.

Pathology. Trachoma is caused by a Bedsonian organism belonging to the psittacosis-lymphogranuloma group—the *Chlamydia trachomatis*; such organisms lie between bacteria and viruses, sharing some of the properties of both. It is seen typically in conjunctival scrapings in colony form in the epithelial cells as Halberstædter-Prowazek inclusion bodies (Fig. 142). The inclusion is first composed of numerous initial bodies. These divide until eventually the cell becomes filled with innumerable elementary bodies embedded in a carbohydrate matrix to form the inclusion body; the nucleus of the cell is displaced to one side and degenerates until eventually the cell bursts and the elementary bodies are set free to attack fresh cells in the cytoplasm of which they increase

FIG. 142. Conjunctival epithelial cell in trachoma showing degeneration of the nucleus and an inclusion body (Lindner).



in size to form initial bodies. Morphologically identical inclusions are found in inclusion conjunctivitis so that although they are not histologically pathognomonic of trachoma, the finding of such bodies in scrapings from the conjunctival epithelium is of considerable diagnostic importance. An infection confers little immunity since re-infection is relatively common; its occurrence has been proved experimentally but since subsequent re-infections are progressively less acute it is probable that some degree of immunity develops.

Histologically there is lymphocytic infiltration involving the whole adenoid layer of the parts of the conjunctiva affected. Special aggregations of lymphocytes, without a definite capsule, form follicles which are generally indistinguishable from those of follicular conjunctivitis except that they tend to show necrosis and contain large multinucleated cells (*Leber's cells*). In the late stages hyaline and other types of degeneration occur; and in all long-standing cases fibrous tissue forms around the follicles, giving rise to cicatricial bands such as are never formed in follicular conjunctivitis, and are characteristic. The grosser forms of cicatrization, however, are found only in cases which have been complicated by a superimposed infection.

Sequelæ. Apart from the results of pannus and corneal ulceration the most malign effects of trachoma are caused by distortions of the lids. A peculiar drooping of the upper lids owing to dense infiltration is very characteristic giving a sleepy appearance to the patient (*trachomatous ptosis*). There is always some scarring (Fig. 143) and when this is extensive the shape of the lids, especially the upper, is altered, a change aided by the infiltration of the tarsus, causing softening and absorption of its dense fibrous tissue; by the later contraction of the new-formed scar tissue the lids may be turned inwards (entropion), causing the lashes to rub against the cornea often with disastrous effects (*trichiasis*). These gross changes, however, rarely occur unless complicating infections have played a major part in the illness. (See Chap. 31.)

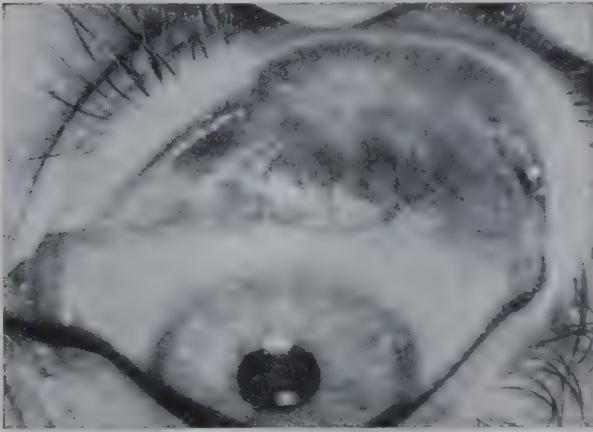


FIG. 143. Trachoma: stage of scarring.

Symptoms. We have already seen that in an uncomplicated trachomatous infection the symptoms may be minimal; when other infections confuse the picture the initial symptomatology depends on these. During the chronic stages there is usually considerable irritation and when the cornea is involved much photophobia and lacrimation, all of which is intensified in the presence of trichiasis. In such cases the disability may be considerable and recurrent irritative phases may result in almost complete incapacity. In endemic areas where trachoma is rife the economic loss thereby entailed may reach enormous dimensions.

Diagnosis. From the clinical point of view, the diagnostic features of trachoma depend on the following characteristics: the presence of follicles, of epithelial keratitis in the early stages most marked in the upper part of the cornea, of pannus in the upper part of the cornea and, in the later stages, of typical trachomatous scarring in the conjunctiva. Depending on the stage of the disease at least two of these signs should be present to establish the diagnosis. It is confirmed by the histological demonstration of the inclusion bodies if inclusion conjunctivitis can be excluded or by the cultivation of the organism.

The disease is frequently designated as occurring in four stages which

were initially suggested by MacCallan, an English ophthalmologist who studied trachoma extensively in Egypt. Trachoma I designates the earliest stages of the disease before clinical diagnosis is possible. Trachoma II includes the period between the appearance of typical trachomatous lesions and the development of scar tissue. Trachoma III is the stage when scarring is obvious. Trachoma IV designates the stage when a cure appears to have been effected or the disease has become quiet but when cicatrization gives rise to symptoms.

Treatment. The most effective treatment particularly in the early stages is either by the sulphonamides (given systemically, reinforced, perhaps, by sodium sulphacetamide drops) or—preferably—by the instillation of one of the wide-spectrum antibiotic drugs such as the tetracyclines. If the disease is complicated by intercurrent infection, the latter treatment should always be given. Much of the dramatic effect which results is due to the elimination of secondary infections which almost invariably flourish in trachomatous eyes, but there is no doubt that the organism disappears and that the histological examination of scrapings from the conjunctiva demonstrates the disintegration of the inclusion bodies after the use of these drugs. It is to be remembered, of course, that well-formed follicles take a considerable time to resolve and dense infiltrative tissue does not disappear so that the full effects of a course of such treatment cannot be assessed for some months. It would seem, however, that some cases are resistant to this treatment and it may be that different strains of the organism exist which have different sensitivities to antibiotic drugs, a variation which occurs in many organismal infections. It is desirable that antibiotic drugs should be instilled several times a day until such time as the disease appears to be inactive.

In the more favourable cases such treatment (combined with mechanical expression of the follicles in florid cases) usually brings about a cure more effectively and more rapidly than the classical and more drastic method of therapy by copper sulphate or silver nitrate. In some cases, blepharospasm has been relieved within twenty-four hours, and inclusion bodies have disappeared in three days: the bulbar conjunctiva becomes white in a few days, pannus is reduced, and corneal ulcers may be healed in a week. In established cases, however, the pathological infiltration is slow to disappear. Relapses may occur in which case a further course of treatment is necessary.

The classical treatment consisted of everting the lids and scouring the conjunctiva with a smooth crystal of copper sulphate fixed in a wooden holder and pointed at the end. It is useless to apply copper gently. The treatment, even under local analgesia, is very painful; and it must be repeated over and over again. Fortunately it is not often required today. But if antibiotic treatment fails to show a response the application of copper sulphate or silver nitrate may be advisable but need be used sparingly and with much less rigour than used to be necessary when reliance had to be placed on them alone.

When the follicles are numerous and very prominent the treatment is shortened by attacking them mechanically. This may be done by

various forms of scarification or expression. The conjunctiva is first thoroughly anæsthetized. Scarification may be performed by a knife or sharp spoon or the follicles may be destroyed by diathermy.

If the follicles in the upper fornix are very large and closely packed it is well to commence treatment by *excising the fornix*. There is always a redundancy of tissue here, and no evil results ensue. The upper lid is doubly everted (p. 98) so as to expose completely the retrotarsal fold. A silk suture is then passed through the fold at each end. By dragging on the sutures the whole fold is drawn out; it is then excised with scissors. If the tarsal plate is much diseased or distorted it also may be excised in the operation of *tarsectomy* which frequently gives much relief in chronic cases.

Pannus requires no special treatment for it quietens with the recession of the conjunctival activity. Corneal ulcers must be treated on general principles (*q.v.*).

Tuberculosis of the Conjunctiva occurs typically in young people who are often free of clinical signs of tuberculosis elsewhere in the body, in which case it is a primary infection of exogenous origin. The disease is rare: it nearly always produces ulceration. Conjunctival ulceration should always suggest either the presence of an embedded foreign body or a tuberculous or syphilitic lesion.

Tuberculosis occurs in several forms: (1) small miliary ulcers usually on the palpebral conjunctiva; (2) granules on the palpebral conjunctiva resembling trachoma follicles; (3) gelatinous cockscomb-like excrescences in the fornices; (4) polypoid pedunculated outgrowths; (5) a solitary nodule near the limbus or on the bulbar conjunctiva, which may become infected with pyogenic organisms and ulcerate. Occasionally the conjunctiva may be affected by extension of lupus vulgaris from the face.

The disease is chronic, and the ulcers are indolent. The preauricular lymph node is often enlarged and may suppurate, but there is little pain or irritation unless the ulceration is extensive.

Pathology. Scrapings may show tubercle bacilli and sections typical giant-cell systems, while inoculation experiments may be made by the intraperitoneal inoculation of guinea-pigs.

Treatment. If the disease is a primary focus, it should preferably be eradicated by excision of the affected conjunctiva; if this is not feasible it should be thoroughly scraped and cauterized by diathermy. In all cases antibiotic treatment with streptomycin topically and systemically combined with PAS or Isoniazid should be administered (Chap. 14). Cases of lupus frequently respond to calciferol.

Sarcoid. Patients affected with sarcoidosis not infrequently show lesions in the conjunctiva. These typically take a nodular form, translucent and orange in appearance, located usually in the folds of the lower fornix. Sometimes they are large and confluent. Diagnosis is by biopsy, and treatment is directed to the general condition.

Syphilis manifests itself rarely in the conjunctiva in the form of a primary chancre which is less indurated than the ordinary genital

chancre and is usually conveyed by an infected mouth. A chronic ulcer or gummatous ulceration of the palpebral or still more of the bulbar conjunctiva is suggestive of the condition particularly when the regional lymph nodes are enlarged. Scrapings should be taken and examined for spirochaetes. A primary chancre of the palpebral conjunctiva may be wrongly diagnosed and treated as a chalazion (*q.v.*).

Tularemia. Tularemia is a disease with a widespread distribution in America, Europe and Asia caused by an organism (the *Brucella tularensis*) derived from animals, particularly squirrels and rabbits. In the oculo-glandular form ulcers and nodules appear on the tarsal conjunctiva associated with swelling of the pre-auricular lymph node and accompanied by general constitutional symptoms of fever and debility. The diagnosis is made by an agglutination test and treatment, locally and systemically, is by streptomycin.

Ophthalmia nodosa is a nodular conjunctivitis which may be mistaken for tuberculosis due to the irritation of the hairs of certain caterpillars, and therefore always commences in the summer months. Small semi-translucent, reddish, or yellowish grey nodules are formed in the conjunctiva, the cornea, and sometimes in the iris. On microscopic examination hairs surrounded by giant cells and lymphocytes are found. The nodules in the conjunctiva should be excised; otherwise the condition is treated on general principles.

Parinaud's oculo-glandular syndrome is a generic name which used to be applied to conjunctivitis, usually of the follicular type, associated with pre-auricular (or sub-maxillary) lymphadenopathy. The cause varies. Infection by a leptothrix or other fungi explains some cases; others may be due to organismal infections such as lymphogranuloma venereum or to the infections already discussed, while others clinically similar are of unknown origin.

A number of syndromes related to **erythema multiforme** affect the conjunctiva in association with other mucous membranes such as those of the mouth, the nose, the urethra and vulva, as well as eruptions on the skin associated with general toxic symptoms sometimes of considerable severity. The conjunctivitis may be mild but sometimes is severe when the condition is often known as *the Stevens-Johnson syndrome*; it may be of a pseudo-membranous or vesicular type and blindness may result from corneal complications. In **REITER'S DISEASE** an acute conjunctivitis, catarrhal or purulent, is associated with urethritis and polyarthritis; a keratitis and a uveitis, sometimes with hypopyon, occasionally occur. The conjunctivitis associated with **BEHÇET'S DISEASE** (*q.v.*) is usually mild. Treatment of all these conditions is unsatisfactory. Corticosteroids and antibiotics (Aureomycin) do not give consistent results.

Benign Mucous Membrane Pemphigoid is a rare but very serious disease of unknown origin affecting both eyes. Vesicles occur on the conjunctiva, but more commonly greyish white membranous patches are seen. Progressive cicatrization follows, leading eventually to *essential shrinkage of the conjunctiva*, with consequent opacification of the cornea (Fig. 144). Similar lesions may be found in the nose, mouth, palate, pharynx, anus and vagina, and more rarely on the skin. Local treatment, such as transplantation of mucous membrane, is unavailing, as also, indeed, is general treatment.

Somewhat similar lesions may complicate other aphthous affections of the mucous membranes such as *erythema nodosum*, *dermatitis herpetiformis*, *epidermolysis bullosa*, and *hydroa vacciniforme*.

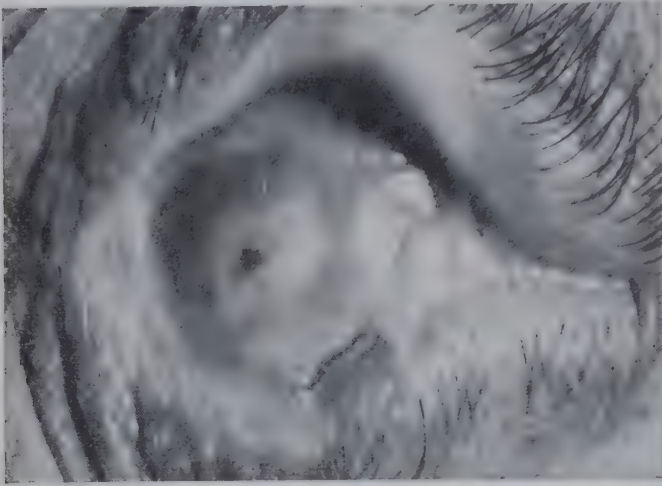


FIG. 144. Pemphigoid of the conjunctiva.

Allergic Types of Conjunctivitis

The allergic reactions of the conjunctiva may assume three forms—an ordinary acute or subacute conjunctivitis of the catarrhal type, phlyctenular conjunctivitis, a characteristic reaction to endogenous allergens, and vernal catarrh, a characteristic reaction to those of exogenous origin.

Acute or Subacute Allergic Catarrhal Conjunctivitis. This type of reaction shows few distinguishing characteristics from the corresponding types of inflammation due to organismal infection except that the hyperæmia is usually marked, the secretion is watery and not purulent but often contains eosinophil cells, and the condition has a chronic habit with a marked tendency to sub-acute remissions on renewed contact with the allergen.

Sometimes the allergen is a bacterial protein of endogenous nature, the most common being a staphylococcus in the nasal cavity or upper respiratory tract. A more characteristic picture is due to exogenous proteins, in which case the conjunctivitis may form part of a typical hay fever. Contact with animals (horses, cats), pollens or certain flowers (primula, etc.) is a frequent cause. Some chemicals, cosmetics and eyelash dyes cause severe conjunctivitis and dermatitis.

Drugs applied locally to the conjunctiva often cause a typical reaction of this type in susceptible persons; it may be violent, almost erysipelatous, in type, spreading widely over the lids and face. The most typical picture of such an acute reaction is that of *atropine irritation*, while eserine and other drugs tend to produce a more chronic response characterized by follicular formation (*q.v.*).

Treatment is logically by removal of the allergen from the environment ; if this cannot be done desensitization may be attempted by a long course of injections. Temporary relief may be obtained by astringent lotions or, more effectively, by the instillation of adrenaline solution (1 in 1,000) or antihistamine drugs (antistine privine, 1.0 per cent.). Corticosteroid drops also frequently bring relief.

In atropine irritation the drug should be avoided. If a mydriatic is imperative, some other should be substituted (p. 40), but frequently in susceptible persons an allergy also develops to these drugs. Lach-esine, which unfortunately is not a strong mydriatic, is particularly useful in this respect. Subconjunctival injections of mydricine (p. 563) may be used with impunity in these cases.

Phlyctenular Conjunctivitis (*Eczematous Conjunctivitis*). In phlyctenular conjunctivitis one or more small, round, grey or yellow

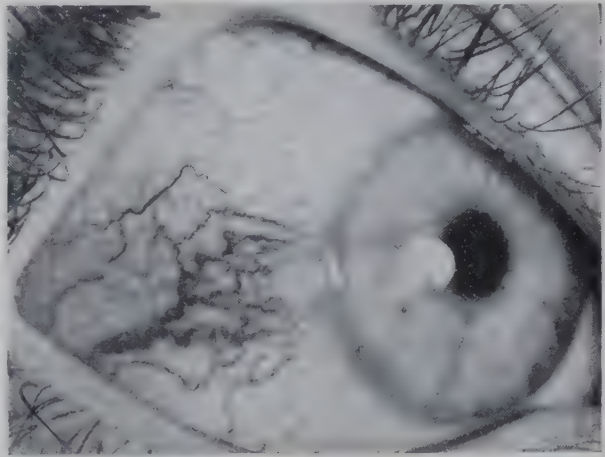


FIG. 145. Phlyctenular conjunctivitis; a phlycten at the limbus.

nodules, slightly raised above the surface, are seen on the bulbar conjunctiva, generally at or near the limbus (Fig. 145) ; they rarely occur on the palpebral conjunctiva. The disease is frequently complicated by muco-purulent conjunctivitis, in which case the whole conjunctiva is intensely reddened. In pure phlyctenular conjunctivitis the congestion of the vessels is limited to the area around the phlyctens.

The disease is most frequent in ill-nourished, weakly children from five or six to ten or twelve years of age and is rare in adults. The children often have enlarged tonsils and adenoids, while lymphatic nodes in the neck or other signs of tuberculosis may be present. The first attack often follows an exanthem, especially measles, and rhinitis and facial eczema are frequently present. As with all other allergic conditions the disease shows a tendency to recur at intervals during the susceptible age-period, particularly when some intercurrent malady leads to the lowering of vitality.

The evidence is considerable that phlyctenular conjunctivitis is

an allergic condition caused by endogenous bacterial proteins which in most cases are tuberculous, but may in other cases be derived from mild infections of long standing as in tonsils or adenoids. The condition used to be very common in Britain and America but is rare today, a change which may in some degree be due to improved hygiene and the control over infection of milk by bovine tuberculosis.

Phlyctens, as the name suggests (*φλύκταινα*, a bleb), at first resemble blebs, but there is a true vesicular stage. They may be so small as to be seen only with difficulty, but they usually measure about 1 mm. in diameter, occasionally reaching a diameter of 3 mm. or 4 mm. ; the larger ones are yellow resembling pustules. In the later stages the epithelium over the surface becomes necrotic and small ulcers are formed ; on the conjunctiva this is of little moment since healing takes place rapidly without the formation of a scar, but when it occurs on the cornea, as is very frequently the case, it is much more serious (p. 208).

Pathology. A simple phlycten shows in section a triangular area of intense infiltration with mononuclear lymphocytes, the apex of the triangle being towards the deeper layers ; initially the epithelium is intact. If there is a considerable amount of conjunctivitis of the muco-purulent type, polymorphonuclear leucocytes are also present, both in the subepithelial tissues and among the epithelial cells ; in these cases the epithelium is quickly desquamated.

Simple phlyctenular conjunctivitis is attended with few symptoms. There is some discomfort and irritation associated with reflex lacrimation. If there is no muco-purulent complication and if the cornea is not involved there is little or no photophobia. Corneal spread, however, is the rule if the condition persists and in this case there is a marked intensification of the symptoms particularly from phlyctens lying astride the limbus.

Treatment. Simple phlyctenular conjunctivitis is usually readily amenable to treatment, which must be both local and general.

Local treatment used to consist in bathing the eyes with a bland lotion and inserting yellow oxide of mercury ointment, 0.5 to 1.0 per cent., within the lids three times a day ; the eye is massaged with a finger placed upon the upper lid, moving the lid upon the globe. As in many allergic conditions, however, steroids given as drops or ointment usually have a most dramatic although temporary effect (Chap. 14). If there is any corneal complication, or evidence of its imminence, atropine is added. If excoriations (rhagades) occur at the outer canthus, they should be treated by touching with the point of the solid silver nitrate stick. The eyes should not be bandaged but dark glasses or a shade, covering both eyes and extending well over the temples, may be ordered.

General treatment is never to be neglected, otherwise recurrence is inevitable. The allergen should, if possible, be eliminated or

desensitization attempted. In the presence of positive skin reactions for tuberculosis, a course of tuberculin should be given ; but it should be used with care since focal reactions may occur in these hypersensitive subjects. Infected tonsils and adenoids should be removed. In addition a general tonic regime should be instigated with abundance of fresh air, good food, sun or artificial light-baths, cod liver oil, vitamins A and D, and any other measures indicated. Calcium in the form of calcium gluconate has been advocated. This general regime must be continued for a prolonged period in order to prevent recurrences, preferably in a bracing and healthy environment ; phlyctenular ophthalmitis should be regarded as a sign of general debility which requires attention.

Spring Catarrh (Vernal Conjunctivitis). This is a recurrent bilateral conjunctivitis occurring with the onset of hot weather, and therefore rather a summer than a spring complaint, found in young people, usually boys. Burning, itching, some photophobia and lacrimation are the chief symptoms accompanied by a characteristic white, ropy secretion. In the cooler months the condition subsides and gives no trouble although the lesions persist, but the symptoms recur with the return of heat. The disease is met with among all classes, is sporadic, and non-contagious. It is probably a hypersensitive reaction to exogenous allergens as indicated by the accompanying eosinophilia.

Two typical forms are seen : (1) the palpebral form ; (2) the bulbar form. Both may be combined, but this is relatively rare.

The *palpebral form* is easily recognized. On everting the upper lid the palpebral conjunctiva is seen to be hypertrophied and mapped out into polygonal raised areas, not unlike cobble stones (Fig. 146). The colour is bluish white, like milk, and this appearance may be seen also over the lower palpebral conjunctiva. The flat-topped nodules are hard, and consist chiefly of dense fibrous tissue, but the epithelium over them is thickened, giving rise



FIG. 146. Spring catarrh : palpebral form.

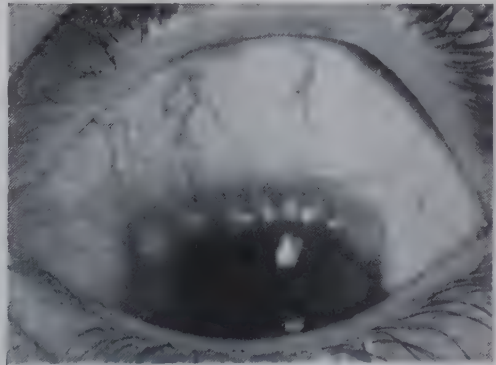


FIG. 147. Spring catarrh : bulbar form.

to the milky hue. Histologically they are hypertrophied papillæ—not follicles. Eosinophilic leucocytes are present in them in great numbers and are found in the secretion. The palpebral form cannot be mistaken if typical, but it may resemble trachoma. The type of patient, the milky hue, the freedom of the fornix from implication and the characteristic recurrence in hot weather will usually prevent mistakes.

The *bulbar form* is less characteristic (Fig. 147). In it nodules or a wall of gelatinous thickening appears at the limbus. Both types are complicated by a fine diffuse superficial punctate keratitis.

Serious complications never supervene and the ultimate prognosis is good, and although recurrences may persist for several years the disease eventually subsides. Occasionally some thickening and discoloration of the conjunctiva may remain.

Treatment is purely symptomatic. The irritation is best relieved by the frequent instillation of steroid drops or ointment; after some days the acute irritation usually subsides and thereafter a maintenance dose three or four times a day during the seasonal period of activity generally keeps the symptoms in check. Weak acetic acid (0.2 to 0.5 per cent.) may give temporary relief, and tinted glasses are often a considerable comfort. In proliferative cases applications of β -radiation at monthly intervals during February, March and April, are sometimes of value in preventing an attack, but do not cure the disease. Excision of the nodules, sometimes advised, is generally useless.

Traumatic conjunctivitis will be considered with ocular injuries when the action of caustics and vesicant gases will be discussed (Chap. 26). It is to be remembered in this connection that malingerers sometimes induce conjunctivitis by the insertion of a multitude of irritants into the eyes. The irritation is most marked in the lower fornix, and usually the right eye is affected in right-handed people.

DEGENERATIVE CHANGES IN THE CONJUNCTIVA

Concretions (“*Lithiasis*”) occur as minute hard yellow spots in the palpebral conjunctiva (Fig. 148). They are due to the accumula-

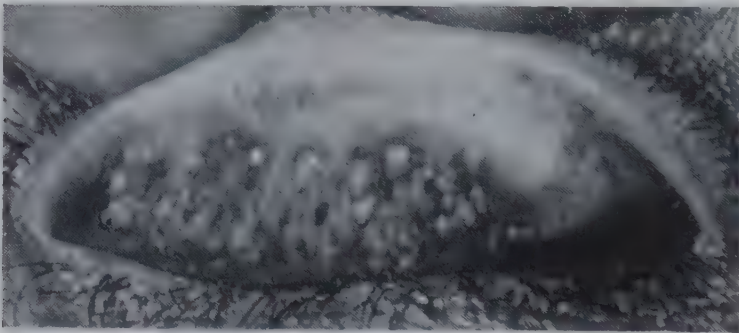
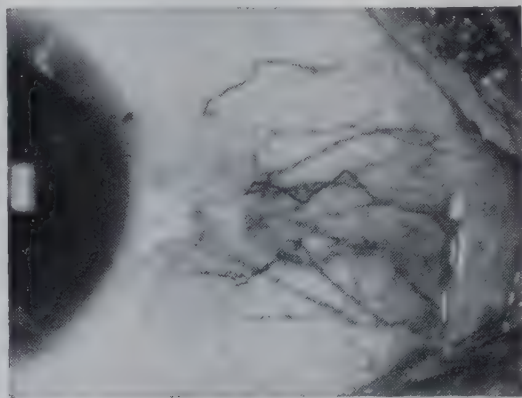


FIG. 148. Concretions in the upper palpebral conjunctiva.

tion of epithelial cells and inspissated mucus in depressions called Henle's glands. They never become calcareous, so the term is a misnomer, but they are so hard that when they project from the surface they scratch the cornea and give the sensation of a foreign body in the eye. They are common in elderly people and should be removed with a sharp needle after cocainization.

Pinguecula is a triangular patch on the conjunctiva, found usually in elderly people, especially those exposed to strong sunlight, dust, wind and so on. It occurs near the limbus in the palpebral aperture, the apex of the triangle being away from the cornea and affects the nasal side first, then the temporal (Fig. 149). It is yellow in colour and looks like fat, whence the name (*pinguis*, fat), but is due to hyaline infiltration and elastotic degeneration of the submucous tissue. Since the pinguecula remains relatively free from congestion

FIG. 149. Pinguecula.



it is particularly conspicuous when the eye is inflamed: mistakes in diagnosis may then occur. It requires no treatment, but may be removed surgically if the disfigurement is great.

Pterygium (πτέρυξ, a wing) (Fig. 150). This is a degenerative condition of the subconjunctival tissues which proliferate as vascularized granulation tissue to invade the cornea, destroying as it does so the superficial layers of the stroma and Bowman's membrane, the whole being covered by conjunctival epithelium. The lesion thus appears as a triangular encroachment of the conjunctiva upon the cornea with, in front of its blunt apex, numerous small opacities lying deeply in the neighbouring part of the cornea. The thick vascularized conjunctiva appears to be drawn onto the cornea from the canthus and is loosely adherent in its whole length to the sclera, the area of adherence being always smaller than its breadth so that there are folds at the upper and lower borders.

A pterygium frequently follows a pinguecula and when single is usually on the nasal side; when double the temporal lesion develops later. In the early stages it is thick and vascular; when it ceases to grow it becomes thin and pale, but never disappears. When it ceases

to progress, consolidation occurs with the formation of dense fibrous tissue and the development of considerable corneal astigmatism. Ordinarily the condition is symptomless, but vision becomes impaired if it progresses into the pupillary area of the cornea.

The condition is common in dry sunny climates with sandy soils as in parts of Australia, South Africa, Texas, or the Middle East. Ultra-violet light may be an aetiological factor.

Treatment. A pterygium is best left alone unless it is progressing towards the pupillary area, or is disfiguring. It cannot be removed without leaving a scar unless it is replaced by a lamellar corneal graft.

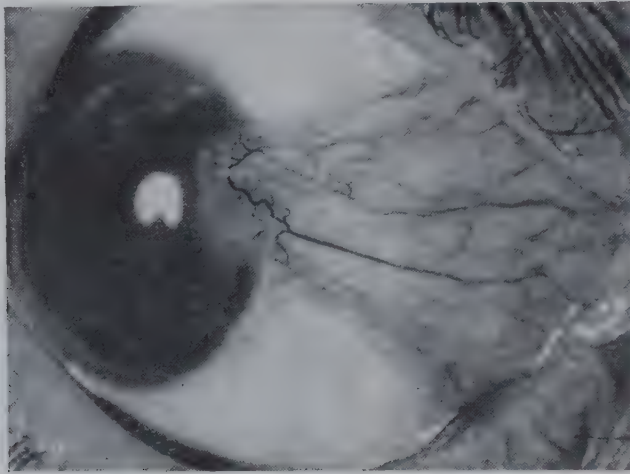


FIG. 150. Pterygium.

Removal is effected by seizing the neck near the corneal margin with fixation forceps, raising it, and shaving or dissecting it from the cornea, starting from the apex. Care must be taken not to go too deeply. The pterygium is then freed from the sclera for about half the distance towards the canthus. Two converging incisions are then made with scissors to excise as much of the pterygium as possible. The conjunctiva is then freed from the sclera above and below so that the two edges can be sutured together.

A pterygium sometimes recurs after removal. This may only be apparent owing to the vascularization of the denuded area. If it actually re-forms and extends towards the pupillary area, the apex should be freed and turned down under the bulbar conjunctiva and sutured in this position (McReynolds). Alternatively the apex may be destroyed by diathermy, or contact radiation by β -rays may be effective.

SYMPTOMATIC CONDITIONS

Subconjunctival Ecchymosis, due to the rupture of small vessels, may be the result of minor injury, or more commonly occurs apparently spontaneously. The condition, though unsightly, is trivial. Very minute ecchymoses, or possibly thromboses, are seen in severe con-

conjunctivitis ; larger extravasations accompany severe straining, especially in old people as on lifting heavy weights or vomiting ; in these circumstances they indicate a weakness of the vessel walls and should suggest the presence of arteriosclerosis for which appropriate steps may be advisedly taken. They are not infrequently seen in children with whooping cough and may occur in scurvy, such blood diseases as purpura, or in malaria ; as a rule they need arouse no anxiety.

More serious are the large subconjunctival ecchymoses which seep forwards from the fornix which sometimes follow injuries when they are due to an extravasation of blood along the floor of the orbit, the result of a fracture of the base of the skull. In fractures of the sphenoid the blood appears later on the temporal side than elsewhere. Hæmorrhages also result from severe or prolonged pressure on the thorax and abdomen, as in persons squeezed in a crowd or by machinery.

Treatment. The blood becomes absorbed in from one to three weeks without treatment ; a bland lotion may be used as a placebo.

Chemosis or œdema of the conjunctiva, may occur in (1) acute inflammations ; (2) in cases of obstruction to the circulation ; (3) in abnormal blood conditions.

In the first group of cases the inflammation may be in the conjunctiva, as in gonorrhœal conjunctivitis, or within the eyeball, as in panophthalmitis or hypopyon ulcer ; it is also occasionally found in acute glaucoma. The inflammation may be in the accessory structures of the eye, as a styne or an insect bite on the lid, dacryocystitis, periostitis, orbital cellulitis or cerebro-spinal meningitis.

In the second group the pressure of an orbital tumour may so interfere with the lymph and blood streams as to produce chemosis ; it is also found in pulsating exophthalmos or exophthalmic ophthalmoplegia.

To the third group belong nephritis and the anæmias, urticaria and angioneurotic œdema.

Xerosis (Xerophthalmia) (ξηρός, dry) is a dry, lustreless condition of the conjunctiva which occurs in two groups of cases : (1) as a sequel of a local ocular affection ; (2) associated with general disease.

The first type is a cicatricial degeneration of the conjunctiva—(a) following trachoma, burns, pemphigoid, diphtheria, etc., commencing in isolated spots, ultimately involving the whole conjunctiva and cornea ; (b) following exposure, due to ectropion or proptosis wherein the eye is not properly covered by the lids.

The chief changes are in the epithelium which becomes epidermoid like that of skin with granular and horny layers and ceases to secrete mucus. A certain amount of vicarious activity is set up in the meibomian glands (Chap. 31) which cover the dry surface with their fatty secretion so that the watery tears then fail to moisten the conjunctiva. The *C. xerosis* grows profusely in these conditions, but this organism has no causal relationship and is of no importance.

It is to be noted that xerosis has nothing to do with any failure of function on the part of the lacrimal apparatus. The conjunctiva can be quite efficiently moistened by its own secretions alone and if the lacrimal gland is extirpated xerosis does not follow. If, on the other hand, the secretory activity of the membrane itself is impaired, xerosis may follow in spite of normal or increased lacrimal secretion.

In the second group of cases xerosis is due to a deficiency of the fat-soluble vitamin A in the diet and occurs usually in a mild form, particularly in children, especially boys, accompanied by night blindness (Chap. 24). It is characterized by small triangular white patches on the outer and inner sides of the cornea, covered by a material resembling dried foam, which is not wetted by the tears (*Bittô's spots*). The foamy spots are due to horny epithelium which is cast off into the conjunctival sac and accumulates in the lower fornix. The cases usually occur during the summer months, and the children are often not conspicuously ill-nourished. A similar mild form, also associated with night blindness, is met with in adults in tropical countries such as India. A severe form (xerophthalmia) is found in marasmic children, associated with keratomalacia and necrosis of the cornea (p. 201).

Treatment. Xerosis is a symptom and its treatment must therefore be purely symptomatic. Local treatment consists in relieving the dryness with methylcellulose, parolein or weak alkaline solutions; dark glasses should be worn. In the deficiency variety restoration of normal nutrition by the administration of vitamin A and other general measures are all-important, although in marasmic cases the patients may be too weak to benefit thereby.

Kerato-conjunctivitis sicca, a condition due to deficiency of the lacrimal secretion, is described elsewhere (p. 508).

Argyrosis is the staining of the conjunctiva a deep brown from prolonged application of silver salts (nitrate, proteinate, etc.) for the treatment of chronic conjunctivitis and especially trachoma. The staining, which is most marked in the lower fornix, is due to the impregnation of the elastic fibres in the membrane and vessel walls with reduced metallic silver. It is very difficult to eliminate but the subconjunctival injection (0.3 ml.) of 2 parts of 2 per cent. potassium ferricyanide with 1 part of 12 per cent. sodium thiosulphate may sometimes be efficacious: a platinum needle must be used in the syringe to avoid staining with iron.

CYSTS AND TUMOURS

The only common **cysts** found in the conjunctiva are due to dilatation of lymph spaces. When small these often form rows of little cysts on the bulbar conjunctiva (*lymphangiectasis*). Occasionally, single though multilocular cysts occur (*lymphangiomata*). Larger retention cysts of Krause's accessory lacrimal glands occur in the upper fornix (Chapter 32). Subconjunctival cysticercus and hydatid cysts are rare. Non-parasitic cysts require simple removal of the anterior walls. *Epithelial implantation cysts* occur rarely after injuries or operations such as tenotomy, and may burrow into the cornea.

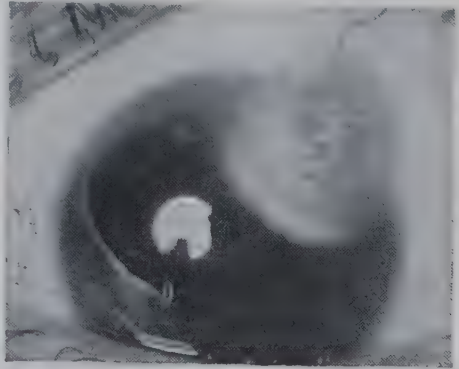
Tumours of the conjunctiva have a tendency to be polypoid owing to the perpetual movements of the globe and lids.

Congenital tumours include dermoids and dermolipomata.

Dermoids are lenticular yellow tumours, usually astride the corneal margin, most commonly at the outer side (Fig. 151). They consist of epidermoid epithelium with sebaceous glands and hairs which may cause irritation. They tend to grow at puberty, and should be dissected off the globe if troublesome, although after removal the site of attachment to the cornea remains densely opaque. This area can be disguised by suitable tattooing or replaced by a lamellar graft.

Dermo-lipomata or *fibro-fatty tumours* are congenital tumours found at the outer canthus sometimes associated with accessory auricles and other congenital defects in babies. They consist of fibrous tissue and fat, sometimes with dermoid tissue on the surface, and are not encapsulated. The main mass should be removed, but it will be found that the fat is continuous with that of the orbit.

FIG. 151. Dermoid cyst.



Papillomata occur at the inner canthus, in the fornices or at the limbus; they may be mistaken for the cockscomb type of tuberculosis. They may become malignant and should be removed.

Simple Granulomata, consisting of exuberant granulation tissue, generally polypoid in form, often grow from tenotomy wounds or the sites of foreign bodies. They are common in empty sockets after excision, and at the site of chalazia which have been insufficiently scraped (p. 490). They should be removed by scissors.

Fibromata, also generally polypoid, occur in sockets. They may be soft or hard, and require simple removal.

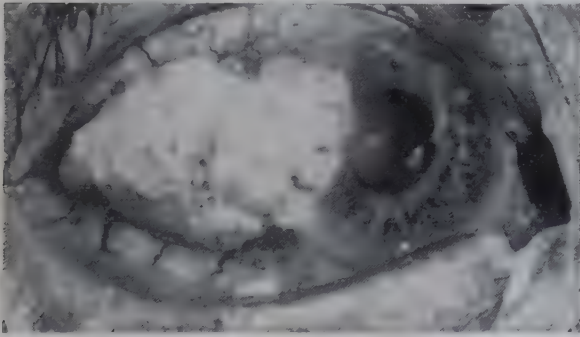


FIG. 152. Epithelioma of the conjunctiva.

Epithelioma (*Squamous-celled Carcinoma*) usually occurs where one kind of epithelium passes into another; in the conjunctiva it therefore occurs chiefly at the limbus (Figs. 152 and 153). *Papillomata* in old people often take on malignant proliferation. *Bowen's intra-epithelial epithelioma* is a rarity. *Epitheliomata* spread over the surface and into the fornices, rarely penetrating the globe. They have the structure characteristic of such growths elsewhere. They must be removed as freely as possible, the base being cauterized by diathermy; the diagnosis should be microscopically confirmed. On the slightest sign of recurrence the

eye must be excised and if recurrence again takes place the orbit must be exenterated and X-ray therapy adopted.

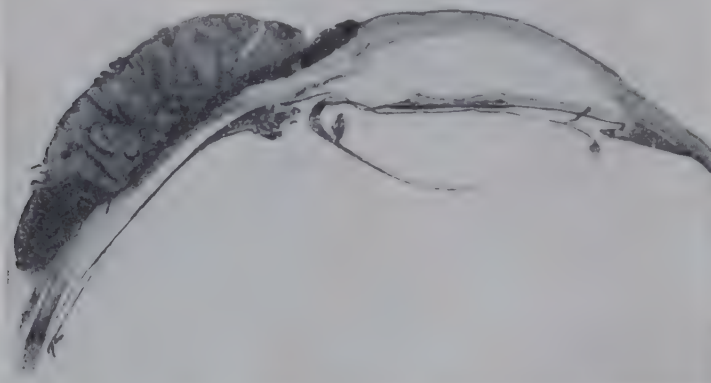


FIG. 153. Epithelioma of the conjunctiva, from a section ($\times 6$).

PIGMENTED TUMOURS constitute an important type of neoplasia which introduces difficult clinical decisions : some are simple (nævus), some potentially pre-cancerous (junctional nævus, pre-cancerous melanosis, lentigo malignum) and some frankly malignant (malignant melanoma).

Nævi or *congenital moles* are not uncommon (Fig. 154). They are grey gelatinous or pigmented nodules situated by preference at the limbus or

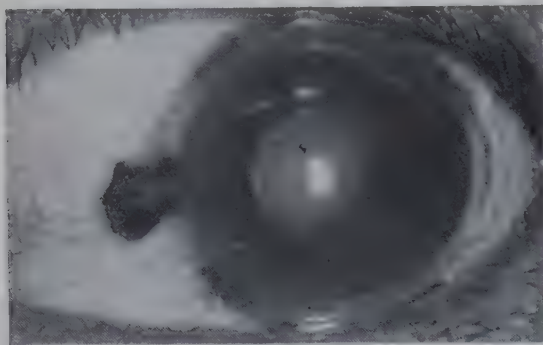


FIG. 154. Nævus of the conjunctiva.

near the plica semilunaris. They have the same structure as in the skin—groups, often alveolar, of “nævus cells” in close connection with the epithelium. They are congenital and tend to grow at puberty, rarely becoming malignant. In view of this they should be excised before puberty, but lest malignant changes follow the operative disturbance, they should always be excised completely. It is to be noted that pigmentation at the limbus occurs normally in dark races, and patches in this situation are not uncommon in people with dark complexions.

Pre-cancerous melanosis is a diffusely spreading pigmentation of the conjunctiva occurring rarely in elderly people, which may also involve the skin of the lids and cheek. It is liable to spread slowly and may eventually assume malignant characteristics, giving rise to metastases.

The condition should therefore always be viewed as pre-cancerous and at this stage it is radio-sensitive ; if allowed to progress to the malignant phase some cases tend to become radio-resistant in which case the only effective treatment is wide excision with exenteration of the orbit and extensive reconstitution by skin grafting.

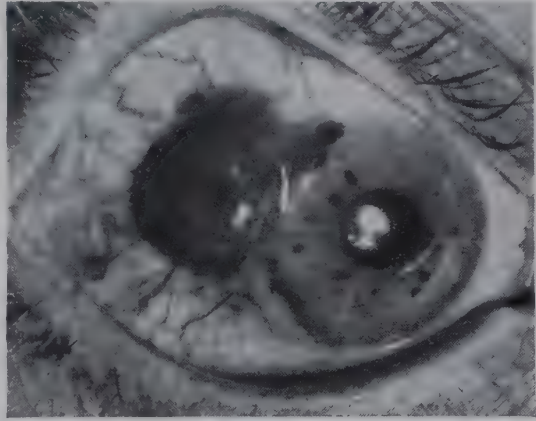


FIG. 155. Malignant melanoma of the limbus.

Malignant melanoma is rare (Fig. 155). It occurs typically at the limbus, is usually pigmented, and most of the patients are old. It spreads over the surface of the globe but rarely penetrates it ; recurrences and metastases occur as elsewhere in the body. The neoplasms may be alveolar—derived from nævi—or round or spindle-celled. The treatment is by excision of the globe or ex-enteration of the orbit.

Rodent ulcer (*Basal-celled Carcinoma*) may invade the conjunctiva from the lids (p. 502).

CHAPTER 16

DISEASES OF THE CORNEA

THE special importance of diseases of the cornea depends upon the fact that they often leave permanent opacities which seriously lower the visual acuity, while the complications which not infrequently attend them may lead to the loss of the eye.

INFLAMMATION OF THE CORNEA (KERATITIS)

Inflammation of the cornea may arise from three sources. (1) *Exogenous infections.* In these cases the cornea is primarily affected, virulent organisms frequently already present in the conjunctival sac gaining access to the corneal tissues.

(2) *From the ocular tissues.* As we have already noted (p. 3), owing to direct anatomical continuity, diseases of the conjunctiva readily spread to the corneal epithelium, those of the sclera to the stroma, and of the uveal tract to the endothelium.

(3) *Endogenous infections.* These are of rare occurrence and, owing to the avascularity of the cornea which allows immunological changes to persist for an unusually long time, they are typically allergic in nature.

From the clinical point of view, corneal inflammations are best divided into two categories, superficial and deep. The former may be purulent (corneal ulcers) or non-purulent.

Superficial Types of Keratitis

Purulent Keratitis, Ulceration of the Cornea. Purulent keratitis is nearly always exogenous, due to pyogenic organisms which invade the cornea from without. It has been pointed out that the only organisms which are known to be able to invade normal epithelium are those of gonorrhœa and diphtheria ; but many other bacteria are capable of producing ulceration, notably the pneumococcus, when the epithelium is damaged. We shall see presently that organisms such as the staphylococcus may cause superficial punctate erosions (*q.v.*).

When we remember the exposed position of the cornea it is not surprising that minute abrasions are extremely common. Although they are probably of everyday occurrence, pathogenic organisms of high virulence are not always present in the conjunctival sac, and if they are, the resistance of the normal tissues may suffice to deal with them. Apart from actual abrasions a diminished resistance of the epithelium will allow the ready intrusion of organisms and

lead to rapid and widespread ulceration into the corneal tissues—drying, as in xerosis, necrosis due to deficient nutrition, as in keratomalacia, desquamation as the result of œdema, neuroparalytic keratitis or general malnutrition. If cocaine is instilled too freely, especially if the lids are not kept closed in the intervals, the epithelium becomes dull and is finally thrown off. Hence this drug is to be used with caution and only under supervision; it should not be given in lotions or drops for continued use.

In the commonest form of suppurative keratitis—the corneal ulcer—there is localized necrosis in the most anterior layers of the cornea. The sequestrum is partly disintegrated and cast off into the

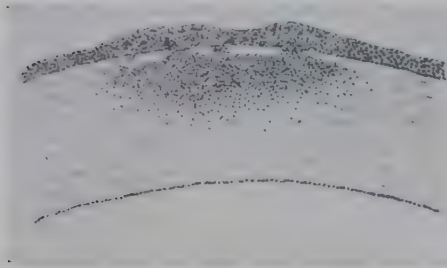


FIG. 156.

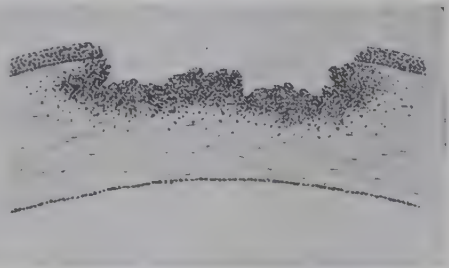


FIG. 157.

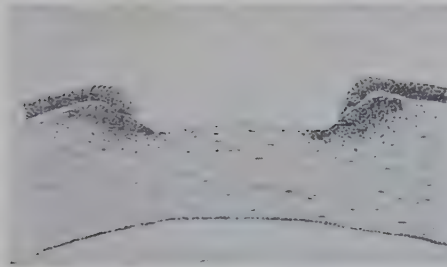


FIG. 158.

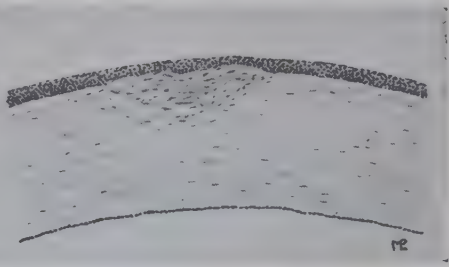


FIG. 159.

FIGS. 156–159. The progressive stages of a corneal ulcer.

conjunctival sac, and partly adheres to the surface of the ulcer. Usually the epithelium is desquamated over an area considerably larger than the ulcer itself, and the same applies to Bowman's membrane. The epithelium, however, rapidly advances towards the ulcer, grows over its edge, and even over the slough or purulent infiltration which forms the floor (Figs. 156–159).

The ulcer is usually saucer-shaped, and the walls project above the normal surface of the cornea owing to swelling caused by the imbibition of fluid by the corneal lamellæ (Fig. 157). The surrounding area is packed with leucocytes, appearing as a grey zone of infiltration. This is the progressive stage.

A line of demarcation forms as in necrosis elsewhere in the body. Here a wall of polymorphonuclear leucocytes forms a second line of

defence while the leucocytes exert their digestive functions, macerating and dissolving the necrotic tissues. When the dead material has been thrown off the ulcer is somewhat larger, but the cloudiness has disappeared, the floor and edges are smooth and transparent, and the regressive stage is reached.

Meanwhile vascularization has been developing (Plate VI, Fig. 1). Minute superficial vessels grow in from the limbus near the ulcer supplying the pabulum to restore the loss of substance; they also supply antibodies and therefore play an important role in combating bacterial infection. Sometimes they are so exuberant as to overstep the limits of utility, as in fascicular ulcer (*q.v.*).

While these events occur in the cornea irritative signs are always found within the eye. Some of the toxins elaborated by the bacteria diffuse through the cornea into the anterior chamber, just as atropine does when instilled into the conjunctival sac. Here they exert an irritative effect upon the vessels of the iris and ciliary body, so that hyperæmia of the iris occurs with or without ciliary injection. The irritation may be so great that leucocytosis takes place, and polymorphonuclear cells, poured out by the vessels, pass into the aqueous and gravitate to the bottom of the anterior chamber where they form a *hypopyon* (p. 195).

The *symptoms* of a corneal ulcer are marked. During the progressive stage there is lachrimation, photophobia, blepharospasm, and pain, owing to the exposure of the terminal fibrils of the ophthalmic division of the fifth nerve.

The term photophobia ($\phi\omega\varsigma$, light; $\phi\acute{o}\beta\omicron\varsigma$, fear; dread of light) is a misnomer. It is the term applied to the blepharospasm set up by corneal irritation, which becomes greatly increased on the slightest attempt to separate the lids, especially if the attempt is made in bright light. This blepharospasm is not abolished in the dark, but is abolished by thorough cocaineization. It is thus a reflex involving the fifth nerve, not the optic nerve. Serious photophobia only accompanies denudation of the epithelium; but spasm of the sphincter of the iris probably increases the discomfort.

The Healing of a Corneal Ulcer. When the ulcer has become vascularized, everything is prepared for cicatrization, which is carried out by the formation of fibrous tissue. The new fibres are not arranged regularly as in the normal lamellæ, so that they refract the light irregularly; the scar is, therefore, more or less opaque. If it is large and dense some of the larger vessels persist; the smaller ones disappear. Bowman's membrane is never regenerated, and if it has been destroyed, as is the case in all but very superficial abrasions, some degree of permanent opacity remains.

The scar tissue which replaces the destroyed portions of the cornea usually fills in the gap exactly, so that the surface is level. It is quite common, however, for some deficiency to remain so that although the resultant cicatrix may be almost transparent, the surface is flattened or even indented. Such *corneal facets* can only

KERATITIS



FIG. 1. Marginal ulcer.



FIG. 2. Hypopyon ulcer.



FIG. 3. Interstitial keratitis.

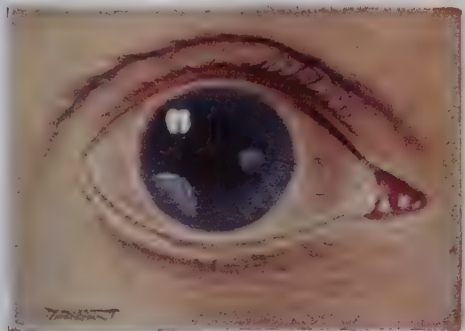


FIG. 4. Corneal nebulae.

SCLERAL DISEASES



FIG. 1. Episcleritis.



FIG. 2. Scleritis.

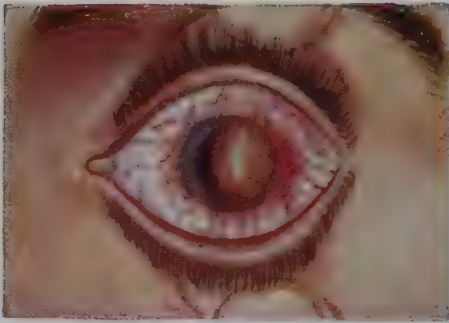


FIG. 3. Tuberculous sclero-keratitis.



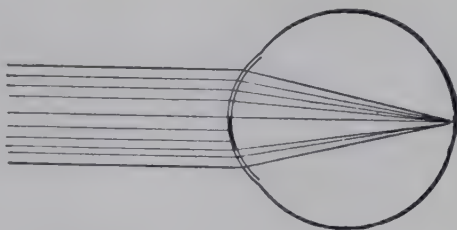
FIG. 4. Ciliary staphyloma.

be seen by careful examination of the corneal reflex (p. 101) but they may cause considerable diminution of the visual acuity.

If the corneal scar is thin the resulting opacity is slight and is called a *nebula* (Plate VI, Fig. 4) ; if rather more dense it is sometimes called a *macula* ; if very dense and white it is called a *leucoma*. Old central leucomata sometimes show a horizontal pigmented line in the palpebral aperture, the nature of which is obscure. A thin, diffuse nebula covering the pupillary area interferes more with vision than a strictly localized dense leucoma, so long as the latter does not block the whole pupillary area.

The reason is that the leucoma stops all the light which falls upon it (Fig. 160), whereas the nebula refracts it irregularly, allowing many of the rays to fall upon the retina where they blur the image formed by the regularly refracted rays. An opacity does not necessarily prevent the light from being focused upon the retina immediately behind it. Thus,

FIG. 160. The optical effect of a complete leucoma. Light falling upon it is not irregularly refracted and does not distort the retinal image.



a central opacity will not prevent the focusing of an object upon the macular region, for the rays passing through the clear peripheral parts of the cornea will be refracted towards the macula ; only those rays which are incident to the corneal surface at the opaque region are cut off. There is thus a loss of brightness rather than of definition, although this is impaired by the superimposition of a diffuse entoptic image of the opacity upon the clear image of the external object.

Although some opacity always remains when Bowman's membrane has been destroyed, there is usually a considerable degree of clearing eventually, a process more marked in younger patients. In this, vascularization plays a considerable part, as is shown by the fact that the opacities clear first in the immediate vicinity of the vessels.

Complications. This simple progress of superficial ulceration and subsequent healing is not unfortunately of universal occurrence. If the ulcer has been deep the loss of tissue may lead to a marked thinning of the entire cornea at the site of the ulcer so that it bulges under the influence of the normal intra-ocular pressure. As the cicatrix becomes consolidated the bulging may disappear, or it may remain permanently as an *ectatic cicatrix* (keratectasia from ulcer).

Some ulcers, especially those due to the pneumococcus and septic organisms, extend rapidly in depth so that the whole thickness of the cornea except Descemet's membrane and a few corneal lamellæ may be destroyed. Descemet's membrane, like other

elastic membranes, offers great resistance to inflammatory processes. It is, however, unable alone to support the intra-ocular pressure : it therefore herniates through the ulcer as a transparent vesicle, called a *keratocele* or *descemetocoele*. This may persist, surrounded by a white cicatricial ring, or it may eventually rupture.

Perforation and its effects. The perforation of an ulcer is usually caused by some sudden exertion on the part of the patient, such as coughing, sneezing, straining at stool, or spasm of the orbicularis. Any such sudden exertion causes a rise in general blood pressure, which at once manifests itself by a rise in intra-ocular pressure and the weak floor of the ulcer, unable to support the sudden strain, gives way. When an ulcer perforates the aqueous suddenly escapes and the intra-ocular pressure sinks to the atmospheric level, the iris and lens being driven forwards into contact with the back of the cornea. The effect upon the nutrition of the cornea is good : owing to the diminution of intra-ocular pressure the diffusion of fluid through the cornea is facilitated, extension of the ulceration usually ceases, pain is alleviated, and cicatrization proceeds rapidly. The complications which attend perforation are, however, of extreme danger to sight and even to the preservation of the eye. These complications vary according to the position and size of the perforation.

Usually the perforation takes place opposite some part of the iris which is washed into the aperture when the aqueous escapes. If the perforation is small the iris becomes gummed down to the opening, the adhesion organizes and an *anterior synechia* is formed. The blocking of the perforation with iris allows the anterior chamber to be re-formed, fresh aqueous being rapidly secreted. If the perforation is large, a portion of the iris is carried not only into the opening but through it, and a *prolapse of iris* is produced. If this does not include the pupillary margin, the prolapse is hemispherical ; if it does, a tag of iris lies free upon the cornea. In either case the

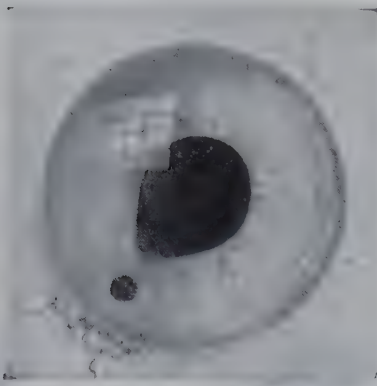


FIG. 161. Perforated corneal ulcer with a prolapse of the iris.

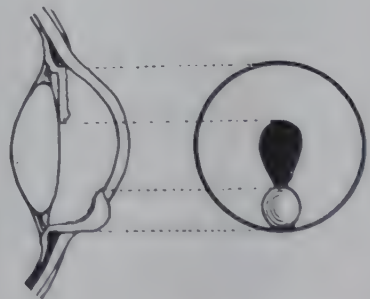


FIG. 162. A diagrammatic view of the topography of the lesion.

colour of the iris soon becomes obscured by the deposition of grey or yellow exudate upon the surface, but eventually the stroma becomes thinned and the black retinal pigmentary epithelium is thrown into relief (Figs. 161 and 162).

Sometimes the whole cornea sloughs with the exception of a narrow rim at the margin, and *total prolapse of iris* occurs. The pupil usually becomes blocked with exudate, and a false cornea is formed consisting of iris covered by exudate. If, however, the perforation takes place suddenly the suspensory ligament of the lens is stretched or ruptured, causing dislocation of the lens, or even its expulsion through the perforation.

If prolapse of the iris has occurred cicatrization may still progress. The exudate which covers the prolapse becomes organized and forms a thin layer of connective tissue over which the conjunctival or corneal epithelium rapidly grows. The contraction of the bands of fibrous tissue tends to flatten the protruding prolapse or *pseudo-cornea*. It rarely, however, becomes quite flat; more commonly the iris and cicatricial tissue are too weak to support the restored intra-ocular pressure, which is often increased owing to the development of a secondary glaucoma (p. 285). The cicatrix therefore tends to become ectatic; such an ectatic cicatrix in which the iris is incarcerated is called an *anterior staphyloma* (Fig. 163) which, depending on its extent, may be either partial or total. The bands of scar tissue on the staphyloma vary in breadth and thickness, producing a lobulated surface often blackened with pigment; hence the name (*σταφυλή*, a bunch of grapes).

If the perforation happens to be opposite the pupil, the pupillary margin of the iris often becomes adherent to the edges and the aperture becomes filled with exudate. The anterior chamber is then re-formed very slowly; the lens remains long in contact with the ulcer, and a permanent opacity may occur in it—*anterior capsular cataract*. As the anterior chamber re-forms, the exudate filling the opening is submitted to strain and frequently ruptures, especially if the patient is restless. This process may be repeated so that the opening may become permanent—*corneal fistula*.

The sudden reduction of intra-ocular pressure when the perforation occurs removes support from all the intra-ocular blood vessels which become dilated and may rupture, causing an *intra-ocular*

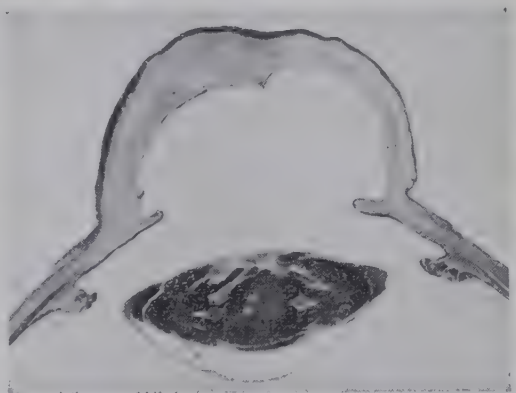


FIG. 163. Anterior staphyloma following the perforation of a hypopyon ulcer (Ashton).

hæmorrhage. Rupture of the retinal vessels gives rise to a vitreous hæmorrhage ; of the choroidal, to a sub-retinal or sub-choroidal hæmorrhage. It may indeed be so profuse that the contents of the globe are extruded with the out-flowing blood.

Finally, the organisms which have caused the ulceration of the cornea may gain access to the interior of the eye as the result of perforation. *Purulent iridocyclitis* or even *panophthalmitis* may thus be set up, a result especially prone to occur in gonorrhœal ophthalmia and in hypopyon ulcer (*q.v.*).

Treatment of Uncomplicated Ulcers. Control of infection, cleanliness, heat, rest, and protection are the fundamental principles of the treatment of corneal ulcers. Control of infection is attained by the use of bacteriostatic drugs : surgical cleanliness is the principle which should regulate the use of lotions ; heat is employed to prevent stasis and encourage repair ; local rest is attained by the use of atropine ; rest and protection from deleterious external agencies are attained by the use of a pad and bandage.

The ordinary treatment of a simple uncomplicated ulcer is as follows. The conjunctival sac is washed out carefully three or more times a day with a considerable quantity of a mild, warm antiseptic lotion. This, as has been noted, acts not as an antiseptic but by washing away secretions and necrotic material, which carry with them many of the organisms and their toxins.

The infection is controlled by the intensive local use of antibiotic drugs as already indicated (Chapter 14). Atropine either as drops or ointment (1 per cent.) should be introduced between the lids two or three times a day ; this drug gives comfort by preventing ciliary spasm and forestalling most of the dangerous results of the iritis which always complicate corneal ulceration. Much comfort can frequently be obtained by hot bathings or compresses (p. 564) and in the intervals between treatment a protective pad and bandage may be applied unless there is much conjunctival discharge ; in this event any benefit is more than counteracted by the retention of secretion and the pad should be replaced by a shade.

If the reaction is severe, steroids administered either as drops or an ointment may control the symptoms dramatically, but should only be used when it is apparent that antibiotics are controlling the infection, and once the inflammation ceases to progress they should be discontinued since they may retard epithelialization and inhibit repair by fibrosis.

To restrain children from touching their bandages a cylinder of stout corrugated cardboard may be applied around the arm, reaching a little beyond the elbow, thus preventing flexion.

If the ulcer progresses despite these therapeutic measures, the removal of necrotic material may be hastened by scraping the floor with a spatula, or the ulcer may be cauterized.

Cauterization may be performed with pure carbolic acid or trichloroacetic acid (10–20 per cent.) or with the galvano-cautery ; the last method is rarely required. Apart from the dangers attending the use of the actual cautery, carbolic acid has the advantage of penetrating a little more deeply than it is actually applied, thus extending its antiseptic properties more widely ; it acts both as a caustic and an antiseptic. No harm is done even if the acid spreads over the normal cornea. Although the parts touched immediately become white, the normal epithelium rapidly recovers. The acid must not, however, touch the conjunctiva lest adhesions form between the lids and globe.

Pure carbolic acid is applied as follows. The patient is seated or lying upon a couch. The ulcer is first stained with fluorescein (2 per cent.), so that its limits may be clearly defined. The conjunctival sac is thoroughly anæsthetized (p. 405). The surgeon separates the lids, scrapes the ulcer with a spatula, and dries it and the surrounding cornea with a piece of blotting paper cut to a point. A wooden match with the end pointed is dipped into the carbolic acid, care being taken to ensure that the wood is thoroughly wet but has no drop of acid hanging from it which may run over the cornea. The ulcer is then touched over the whole of its surface with the point of the match, whereupon it becomes white.

Cauterization with carbolic acid may be repeated two or three times at intervals of one or two days if the ulcer still progresses. If this treatment, combined with the use of antibiotics, atropine, and hot bathings, does not check progress, the actual cautery may be used ; in desperate cases the centre of the floor of the ulcer may be perforated by the cautery to allow the aqueous to escape and establish better conditions of nutrition.

If despite these measures ulceration progresses, pain continues and perforation seems imminent, the latter event may sometimes be anticipated with advantage by *paracentesis*. By this procedure the aqueous is evacuated slowly and the more dangerous results of spontaneous perforation may be avoided while, as has already been pointed out, the nutrition of the diseased cornea is considerably improved. Such an operation, the technique of which will be described subsequently (Chapter 27), may be performed through the floor of the ulcer or just inside the periphery of the cornea.

Any contributory cause of ulceration must, of course, receive attention. Prominent among such causes are conjunctival conditions and general malnutrition. Thus, the ulcers associated with trachomatous pannus will not heal if the lids are neglected. Moreover if the lacrimal sac is infected, this condition must be urgently treated (p. 512). In debilitated adults or old people and marasmic children the building up of the constitution by good food, fresh air, and tonics is often more important than the treatment of the local condition. Large doses of ascorbic acid, injected intravenously if necessary, are frequently of considerable value in such cases where the general health is not good.

When cicatrization is complete and all irritative signs have passed off, attempts to render the scar more transparent are usually disappointing.

Cicatrices clear considerably in young patients, and in many others a gratifying improvement may be noticed in the course of some months. Stimulatory treatment with irritants such as insufflations of finely powdered calomel, massage with yellow oxide of mercury ointment or the instillation of dionine is generally without effect. Old, degenerated or calcareous cicatrices are best left alone, since they are liable to break down and form ulcers which refuse to heal owing to the lack of resistance in the scar tissue.

In some cases the scar remains permanently richly vascularized, making the eyes irritable and subject to repeated attacks of inflammation. The topical administration of steroids (Chapter 14) is frequently effective in controlling these distressing symptoms, but their action is often temporary. Radiation with β -rays is sometimes useful. Although uncertain in its results, *peritomy* is sometimes valuable or the re-growth of large new vessels may be prevented by touching them with a diathermy needle.

In the operation of peritomy, a collar of conjunctiva, 5 mm. broad, is excised round the corneal margin. The raw surface of the sclera should be seared with the galvano-cautery, the object being to destroy the vessels and prevent their re-formation.

If a small dense leucoma covers the pupillary area vision may be improved by an optical iridectomy (Chapter 27).

Some improvement in appearance may be obtained by *tattooing* dense leucomata. It is only suitable for firm scars in quiet eyes, and even then is not without danger. More justifiable is the tattooing of small central nebulæ ; it has the effect of cutting off the irregularly refracted rays, so that vision is improved (p. 189).

Tattooing with Indian ink has been replaced by impregnation with gold (brown) or platinum (black) : of these the latter is preferable. The affected area is denuded of epithelium and a piece of blotting-paper of the same size, soaked in a fresh 2 per cent. solution of platinum chloride, is applied. On removal a few drops of fresh hydrazine hydrate (2 per cent.) solution are allowed to flow over the area, which becomes black. The eye is irrigated with saline, a drop of parolein instilled, and a pad and bandage applied. The epithelium grows over the black deposit of platinum ; but at a later stage it may break down.

Keratectomy is a further expedient by which a superficial scar is shaved off and the epithelium allowed to grow over the defect thus formed. A better result follows a *lamellar keratoplasty* (Chapter 27) whereby the defect is filled by a correspondingly shaped graft from a human (cadaver) eye. When the scar traverses the greater part or the whole of the corneal thickness, a *full-thickness (penetrating) graft* is similarly used (Chapter 27).

The Treatment of a Perforated Ulcer. If perforation has occurred the treatment depends upon its size and situation. If it is small and in the pupillary area prolapse of iris is not to be feared. Rest in bed, the continued use of antibiotics, atropine, and a firmly

applied bandage suffice : all forced expiration—blowing the nose, coughing, etc.—must be avoided. A sneeze can often be inhibited by firm pressure with the finger upon the middle of the upper lip close to the nose. If a small perforation is over the iris, adhesion to the cornea usually occurs. This may become detached when the anterior chamber re-forms, or may be drawn out into a fine thread in which case no special treatment is required.

If prolapse of iris has occurred it should usually be abscised (Chapter 27). No attempt should be made to replace the prolapse in these cases because the iris has become soiled with pus and replacement may result in infection of the interior of the eye and panophthalmitis. Abscission of a prolapsed iris is, however, only possible within the first few days, before adhesion has become firm. It is not to be performed after this has occurred, nor in the case of very large prolapses. In the latter there is so large an opening in the cornea that a permanent fistula may result with loss of the eye from diminished tension and shrinking.

In very large prolapses there is much bulging and the base is often constricted. Every attempt should then be directed to obtaining a flat cicatrix. In addition to rest in bed and the treatment already advised, a pressure bandage must be applied for a prolonged period. A pressure bandage differs from an ordinary protective bandage only in that the space around the eye is packed carefully with cotton-wool to the level of the nose and considerable pressure is exerted in applying the bandage.

Keratocele may be treated first by rest and a pressure bandage but lamellar keratoplasty is most effective ; alternatively the vesicle may be punctured and the case treated as a perforated ulcer.

Fistula of the cornea is most effectively treated by its excision and replacement with a full-thickness graft. Alternatively the edges of the fistula may be cauterized with the actual cautery, or touched with silver nitrate—a procedure which should be undertaken only if there is some trace of an anterior chamber, lest the lens be injured. A conjunctival flap is then drawn over the fistula.

Hypopyon Ulcer. We have already seen that every corneal ulcer is associated with some iritis owing to the diffusion into the inner eye of the toxins elaborated by the bacteria ; the resultant iridocyclitis may be so severe and the outpouring of leucocytes from the vessels so great that these cells gravitate to the bottom of the anterior chamber to form a *hypopyon*. Such a hypopyon consists of polymorphonuclear leucocytes which accumulate in the lower angle of the anterior chamber and eventually become enmeshed in a network of fibrin. The hypopyon may be so small that it is scarcely visible, being hidden behind the rim of sclera which overlaps the cornea. It may reach half-way up the iris, having a flat upper surface determined by gravity, or it may fill the anterior

chamber, wholly obscuring the iris. The larger hypopyons are usually less fluid owing to the fibrinous network which imprisons the leucocytes in its meshes ; these are much less readily absorbed and it may be necessary or advisable to evacuate them.

It is important to remember that the hypopyon is sterile since the leucocytosis is due to toxins, not to actual invasion by bacteria which, indeed, are as incapable of passing through the intact Descemet's membrane as are leucocytes. This accounts for the ease and rapidity with which hypopyon is often absorbed : it may develop in an hour or two, rapidly disappear, and as readily reappear. Such hypopyons are fluid, always moving to the lowest part of the anterior chamber if the position of the patient's head is changed. The fact that the hypopyon is sterile has great practical importance—it is unnecessary to remove the pus, as is the rule in other parts of the body ; if the ulcerative process is controlled the hypopyon will be absorbed.

The development of a hypopyon depends on two factors—the virulence of the infecting organism and the resistance of the tissues. Many pyogenic organisms (staphylococci, streptococci, gonococci, *Moraxella*) may produce this result, but by far the most dangerous are *Ps. pyocyanea* and the pneumococcus which is not infrequently present in the normal conjunctival sac and is particularly likely to be present if there is any inflammation of the lacrimal sac (dacryocystitis). The presence of dacryocystitis is therefore a constant menace to the eye. On the other hand, the agent which produces the injury may carry the infection. The commonest causes are scratches with the finger nail and minute foreign bodies, especially of stone or coal in quarries or mines.

Unless the organism be very virulent some lack of resistance on the part of the tissues must be predicated. Hence hypopyon ulcers are much more common in old, debilitated or alcoholic subjects. These ulcers also occur during or after acute infectious diseases, such as measles, scarlet fever or smallpox.

Hypopyon ulcers vary in type according to the infective agent and the age of the patient. In adults the commonest cause is the pneumococcus and the ulcer formed by it is of a characteristic type, called *ulcus serpens* from its tendency to creep over the cornea in a serpiginous fashion.

The typical *ulcus serpens* is a greyish-white or yellowish disc near the centre of the cornea (Plate VI, Fig. 2). The opacity is greater at the edges than at the centre and is particularly well marked in one special direction. A cloudy grey area, made up of fine lines, surrounds the disc, but is also more marked in the same direction. The whole of the cornea may be lustreless or hazy. There is a violent iritis, and the aqueous is cloudy, or there may be a definite hypopyon. The lids are slightly cedematous, and there is conjunctival and ciliary congestion. The subjective symptoms at the early stage are pain in the eye and

brow and a variable amount of photophobia. There is remarkably little pain after the initial stage; hence treatment is often unduly delayed, with disastrous results.

The ulcer increases in size and depth. On the side of the densest infiltration, which often looks like a yellow crescent, the tissues break down and the ulcer spreads (Fig. 164); on the other side it may be undergoing simultaneous cicatrization and the edges may be covered by fresh epithelium. In this manner it travels forwards. Often there is infiltration just anterior to Descemet's membrane at a spot exactly opposite the floor of the ulcer, while the intervening lamellæ are normal. This fact accounts to some extent for the great tendency to perforation, since the inflammatory process is going on as it were from both surfaces of the cornea. Meanwhile the hypopyon has become more evident, but it may vary in size from hour to hour. As might be expected, the intra-ocular pressure is often raised.

If left to pursue its natural course the hypopyon will increase and become fibrinous, the ulcer will perforate, usually forming a large

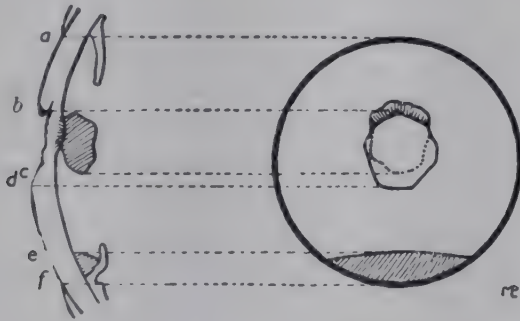


FIG. 164. Hypopyon ulcer. *b-d*, the extent of the ulcer; *b*, actively progressive margin; *b-c*, mass of leucocytes and fibrin adherent to the endothelial surface; *e-f*, hypopyon.

opening through which the iris prolapses. The whole cornea, except the narrow rim nourished by the limbal vascular loops, may necrose, and panophthalmitis destroy the eye. In other cases an extremely dense cicatrix in which the iris is incarcerated (*adherent leucoma*) destroys sight. This may be flat or ectatic (anterior staphyloma). Sometimes the iris is bound down to the lens before perforation occurs. In such cases there are posterior synechiæ, which may be annular or total (p. 230), and the pupil may be occluded by exudates which organize into fibrous tissue.

Treatment. In all cases of hypopyon ulcer the treatment already indicated for corneal ulcers must be initiated at once and must be energetic. After the overhanging edge and the underlying infiltrated tissue have been curetted with a small spud, intensive antibiotic treatment should be instigated at once and when a bacteriological examination has been made and the sensitivities of the organisms determined, it may have to be reassessed. One of the wide-spectrum antibiotics should be instilled hourly or even more often; if there

is a suspicion of the presence of *Ps. pyocyanea*, soframycin should be used. Surface application should be supplemented by the sub-conjunctival injection of this latter drug or of penicillin (p. 142). Atropine is instilled even if the tension is raised (*vide infra*). In many cases the progress of the ulcer and the violence of its clinical symptoms may be controlled by the topical application of steroids (p. 145), *but these should not be employed until the infection appears to be controlled by antibiotics*, lest the inhibition of reparative processes leads to rapid spread or even perforation.

Coincidentally with this treatment the ulcer may be cauterized (p. 193). If this is performed skilfully it does no harm and may save the eye. It is seldom necessary in children.

If these means fail, and especially if the tension of the eye is raised, further measures must be adopted. Of these the most important is *paracentesis*. It may be performed in the standard way (Chapter 27), but in the case of hypopyon ulcers the drastic operation of *Saemisch's section* (*q.v.*) offers some advantages. The latter consists of dividing the ulcer from one side to the other, the ends of the incision being in healthy corneal tissue. If the hypopyon is very fibrinous it may be necessary to pull the coagulum out with the smooth iris forceps (Fig. 305). This procedure improves the nutrition of the cornea and evacuates the hypopyon, but is only to be employed as a last resort; it is rarely necessary if antibiotic treatment has been instituted within a reasonable time.

The commonest cause of failure in treatment is the development of secondary glaucoma, a complication which usually occurs in elderly people. If the tension rises the effect on the cornea is bad for it diminishes the flow of nutrient fluid and therewith its resistance. The rise of tension is not a contra-indication for the exhibition of atropine but is a definite indication for prompt paracentesis or even Saemisch's section if the condition is not rapidly controlled.

If there is a mucocele the lacrimal sac should be excised as soon as possible; if this is not practicable the puncta should be sealed off by diathermy (p. 516).

The *diplobacillary hypopyon ulcer* is best treated with oxytetracycline which should be used topically as 0.5 per cent. ointment (p. 143). An alternative is zinc sulphate solution (0.25–1 per cent.) used every hour or two, and an ichthyol (1.5 per cent.) zinc sulphate (0.5 per cent.) ointment applied to the lids.

Mycotic Hypopyon Ulcer. A rare form of ulcer is due to a fungus such as *Aspergillus fumigatus*. In it the slough is dry in appearance, and is surrounded by a yellow line of demarcation which gradually deepens into a gutter and there may be a hypopyon. Other fungi may be responsible, such as *Candida albicans*. *Treatment* is as for other hypopyon ulcers; the organisms are sensitive to nystatin and amphotericin B.

Marginal Ulcer (Catarrhal Ulcer). Ulcers not infrequently occur near the limbus, especially in old people ; they also occur in association with a chronic conjunctivitis and may be caused by the *Moraxella* or the *H. ægyptius* (Fig. 165). They are shallow, slightly infiltrated and often multiple, and may be accompanied by neuralgic pains in the face and head (Plate VI, Fig. 1). Sometimes they heal rapidly but as rapidly recur, so that the process tends to drag on indefinitely. Frequently the ulcers become vascularized and the vessels persist. More serious rare forms of deep marginal ulceration also occur in old people, constituting a *ring ulcer*, sometimes leading to necrosis of the whole cornea.

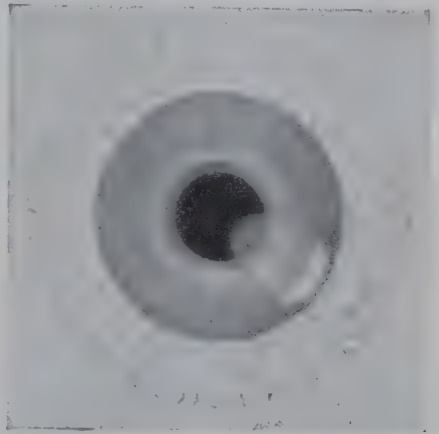


FIG. 165. Catarrhal ulcer.

Treatment. The infection should be treated by an appropriate antibiotic and in all cases any conjunctivitis should be cleared up as far as possible. Mild recurrent marginal ulcers may clear on painting with weak silver nitrate solution, 1 per cent. Steroid drops or ointment may occasionally be of temporary benefit, while the administration of large doses of vitamins B and C may be of value. Recurrence, however, can only be prevented, and that with difficulty, by constitutional treatment which includes appropriate measures to deal with the general condition and the eyes should be protected with dark glasses. If this treatment fails, and the ulcers are vascular, destruction of the vessels with the actual cautery is most likely to succeed. Sometimes (curiously) eserine does good in these cases.

In the deep ring ulcer of old people, silver nitrate or the actual cautery may be used. Paracentesis may avert perforation and improve the nutrition of the cornea. Every attempt should be made to build up the constitution by a nutritious diet and tonics with an abundance of vitamin C.

Chronic Serpiginous Ulcer (Rodent Ulcer, Mooren's Ulcer). This is a rare superficial ulcer of a degenerative type, usually occurring in elderly people, starting at the corneal margin and spreading over the whole of this tissue (Fig. 166). It is accompanied by severe and persistent neuralgic pain and lacrimation. It commences as one or more grey infiltrates ; these break down, forming small ulcers which spread and sooner or later coalesce. The ulcer undermines the epithelium and superficial lamellæ at the advancing border, forming a whitish overhanging edge which is characteristic, while the base becomes quickly vascularized. It rarely perforates, but progresses with intermissions for months until eventually a thin nebula is formed over the

whole cornea and sight is much diminished. In about a quarter of the cases both corneæ are affected, but not always simultaneously. The cause is unknown.

Treatment is extraordinarily difficult. An attempt may be made to apply surgical methods. The overhanging edge should be cut off with scissors, and then the whole surface of the ulcer, and especially the margin, should be well cauterized with the actual cautery, or with trichloroacetic acid (10–20 per cent.), and covered by a conjunctival flap. The tension should be kept low by repeated paracenteses. A few cases have responded to repeated applications of absolute alcohol to the ulcers; to zinc ionization followed by covering with a conjunctival flap; to β -irradiation; or to extensive lamellar grafting; but more commonly treatment fails to stop the process which has even been known to recur in the cicatrized cornea.



FIG. 166. Mooren's ulcer.

Conditions favouring Ulceration. Certain conditions are of considerable importance since they facilitate the development of corneal ulcers and prevent their ready healing. These may be systemic, as in states of profound malnutrition wherein *central marasmic ulcers* or *keratomalacia* occurs. Alternatively the state of the cornea may be unhealthy, due either to the presence of degenerative scars (*atheromatous ulcers*) or to its exposure (*keratitis e lagophthalmo*) or insensitivity (*neuromparalytic keratitis*).

Central Marasmic Ulcer. Symmetrical central ulcers of an indolent type are not infrequently met with in badly nourished children. They may be superficial, show little infiltration and no vascularization forming shallow pits or facets about 2 mm. in diameter with little or no reaction, either in the form of lachrimation or photophobia. Alternatively, they may rapidly perforate, allowing a knuckle of iris to prolapse; this should not be cut off, owing to the relatively large gap in the cornea and the defective powers of repair in the debilitated patient.

Treatment must be directed especially to improving the general nutrition with an abundance of good food, particularly vitamins A and C. Atropine and a bland lotion are used locally.

Keratomalacia, common in countries such as India or in the Far East, affects badly nourished children who are lacking vitamin A, often early in the first year of life ; the condition is usually bilateral. In the rare cases in which the children are old enough to exhibit this symptom the disease commences with night blindness. The conjunctiva becomes dry and shows xerotic spots (p. 182), while the cornea becomes dull and insensitive ; the haze increases and yellow infiltrates form until finally the whole tissue necroses and may seem to melt away within a few hours. A characteristic feature is the absence of inflammatory reaction. The children are usually extremely ill and very frequently die. Owing to their apathetic condition they do not close the lids, so that the cornea is continually exposed.

Treatment must be directed to the general health. Vitamin A should be given in quantity as cod-liver oil, halibut-liver oil or carotene in oil, or in an emergency as an injection. Abundance of fluid in the form of saline should be given, intravenously, subcutaneously or per rectum. The lids should be kept closed under moist warm compresses.

Atheromatous Ulcers occur in old dense leucomata, especially such as have undergone degenerative changes resulting in the formation of hyaline and calcareous deposits. Such scars have little vitality and the deposits act as foreign bodies. They readily succumb to infections, so that when ulceration once begins it proceeds rapidly and deeply with little or no effort at repair ; perforation is often followed by panophthalmitis.

Treatment. The eye is frequently blind and disfiguring ; in such cases it is well to excise it at once, thus relieving the patient of much unnecessary misery. If the eye is worth saving the condition must be treated on general principles.

Keratitis e Lagophthalmo occurs in eyes insufficiently covered by the lids. The epithelium of the exposed cornea becomes desiccated and the substantia propria hazy. Owing to the drying, the epithelium is cast off and the cornea falls a prey to infective organisms.

The condition is due to any cause which may produce exposure of the cornea (lagophthalmos), such as extreme proptosis as in exophthalmic ophthalmoplegia or orbital tumour, paralysis of the orbicularis, and so on. The absence of reflex blinking is an important factor, as well as defective closure of the lids during sleep, so that patients extremely ill from any disease are liable to this form of keratitis.

Treatment consists in keeping the cornea well covered. In mild cases it is sufficient to bandage the eyes at night. If possible the cause of the exposure must be removed, but in the meantime it may be necessary to sew the lids together (p. 414).

Neuroparalytic Keratitis occurs in some cases in which the fifth nerve is paralysed, most typically as a result of the radical treatment

of trigeminal neuralgia. It is relatively rare in nuclear and fascicular lesions within the central nervous system unless the facial nerve is simultaneously involved, but may be caused by intracranial tumours, gummatous basal meningitis, and fractures of the skull. Nor does it occur in all cases of peripheral lesions of the fifth nerve ; thus, if the gasserian ganglion is removed or the fifth nerve injected with alcohol for trigeminal neuralgia, with proper precautions only a small proportion of the cases develops neuroparalytic keratitis, the tendency being decreased if a cervical sympathectomy is performed on the same side.

It is probable that the disease is due to irritative and metabolic changes in or about the degenerating nerves, and that mere section or paralysis of the nerve is unable to produce the disease in the absence of such irritative conditions. Antidromic impulses and axon reflexes play a large part in the local control of the metabolism of the tissues supplied by this nerve, and it is possible that excessive output of histamine-like substances may account for "trophic" skin and other lesions, such as neuroparalytic keratitis. Unawareness of the presence of minute foreign bodies or abrasions by the anæsthetic cornea is another factor. On the other hand, the worsening of the disease by exposure and its amelioration by protection indicate that absence of reflex blinking plays a considerable part in the ætiology, as also, in all probability, does desiccation of the cornea. The reduction of lachrymation in these cases is often striking ; the conjunctiva in most of them stains with rose bengal.

The characteristic feature of neuroparalytic keratitis is the desquamation of the corneal epithelium. The surface of the cornea becomes dull and the epithelium is thrown off, first at the centre, then over the whole surface except a narrow rim at the periphery ; the whole of the epithelium may thus peel off intact. The substantia propria then becomes cloudy and finally yellow, breaking down into a large ulcer which is usually accompanied by a hypopyon. There is no pain, owing to the anæsthesia, but ciliary injection is marked. A large perforation occurs if the case is not speedily treated. In any case, the resulting leucoma is generally so large as to destroy useful vision. Relapses are the rule, the healed scar quickly breaking down again and the whole process being repeated.

Treatment. The ordinary treatment of corneal ulcer should be tried as a preliminary, special care being devoted to the protection of the eye with a bandage. Improvement is often marked, but directly the bandage is relinquished the ulceration starts anew. Closure of the lacrimal puncta to conserve moisture by abolishing the drainage of tears is sometimes of great value. If, however, relapses occur, it is best to suture the lids together (*tarsorrhaphy*) (p. 414) and to keep them sutured for a long period up to at least one year, only separating them when the conditions of the weather—warmth, absence of high winds, and so on—are favourable. In the

operation of tarsorrhaphy after removal or blockage of the gasserian ganglion, no anæsthetic is necessary since sensation is lost in the conjunctiva and lids. The beneficial effect of this procedure is very striking, for it will invariably succeed in stopping the process. Even if a hypopyon ulcer is present cicatrization rapidly takes place and the hypopyon disappears. The keratitis, however, frequently recurs when the lids are separated, even after many months of protection. In the worst cases the eye is useless, and if it tends to become a source of perpetual trouble it is best to excise it.

Non-Suppurative Superficial Keratitis. Superficial non-purulent keratitis includes a number of conditions of varied ætiology. Many of them are viral infections while others such as phlyctenular and rosacea keratitis have a constitutional origin; some of them may lead to the formation of ulcers and pass into the purulent type of keratitis, while in other cases the deeper tissues may be affected and a deep keratitis results.

Infective (Non-suppurative) Superficial Keratitis

Superficial keratitis may result from a number of infections, most of them of the viral type. Of these the most common are herpes, zoster, the adenoviruses and the Chlamydia of trachoma and inclusion conjunctivitis: the last two conditions have already been discussed. More rarely the viruses associated with measles, vaccinia and mumps, as well as the presumptive viruses responsible for Behçet's syndrome and Reiter's syndrome, may affect the cornea, while a secondary keratitis may follow a lid infection with the viruses of molluscum contagiosum and warts (verruçæ). These viral infections give rise to several types of clinical picture, but it is to be remembered that the same appearances may be associated with infection by several types of virus, while one virus may give rise to more than one type of lesion.

Punctate Epithelial Erosions (Multiple Superficial Erosions) are the most common manifestation of viral infections. In this condition the cornea shows multiple minute defects in the epithelium which stain with fluorescein, frequently associated with an acute onset combined with conjunctivitis; there is considerable pain, photophobia and lacrimation. As a rule the infection is characterized by recurrences when fresh erosions appear in successive crops after the initial lesions have quietened or the original erosions have healed; if these recurrences persist for a considerable time, superficial vessels may invade the cornea. A completely non-specific lesion of this type, for example, may be caused by the toxin of the staphylococcus, the organism also giving rise to a blepharitis or conjunctivitis. Some chemical irritants produce the same picture. Associated with a general febrile disturbance this is a well established manifestation of an infection by one or other of the adenoviruses

and, as we have seen, constitutes the characteristic picture of early trachomatous keratitis.

Punctate epithelial keratitis (superficial punctate keratitis) attacks the deeper layers of the corneal epithelium and is sometimes associated with opacities extending into Bowman's membrane and the



FIG. 167. Superficial punctate keratitis.

superficial layers of the stroma (*punctate subepithelial keratitis*). The epithelial opacities appear as superficial slightly raised grey dots scattered over the central area of the cornea which do not stain readily with fluorescein but turn a deep red with rose bengal (Fig. 167). A combination of epithelial and subepithelial punctate lesions is also a common occurrence in viral infections (*epidemic kerato-conjunctivitis, pharyngo-conjunctival fever, herpes, vaccinia* and others (*q.v.*)), but may occur without known cause (*Thygeson's superficial punctate keratitis*).

Treatment of most of these conditions is symptomatic consisting of bland lotions and atropine, and has so far been ineffective; antibiotics are useful in eliminating secondary infections.

Herpes (Simplex). The virus of herpes has a widespread distribution; it can be grown in tissue-culture and the elementary bodies can be found by suitable staining methods in the vesicular fluid. The infection is transferable to the corneæ of rabbits in which animals a herpetic encephalitis may subsequently develop. There is some evidence that the virus has antigenic properties but in practice immunity does not develop after an attack; indeed, a person once infected frequently becomes a carrier, and periodic attacks tend to break out on the lips, nose and genitals as well as on the cornea, the recurrences taking place particularly in association with such intercurrent diseases as a febrile cold, pneumonia or a malarial relapse or even after typhoid inoculation.

In a primary lesion (usually in children) herpes may manifest itself as a severe follicular kerato-conjunctivitis (*q.v.*); but in the usual recurrent forms the conjunctiva is spared apart from the occurrence of a nondescript conjunctivitis. The cornea essentially is involved, the initial lesion being a *superficial punctate keratitis*. In this condition numerous whitish plaques of epithelial cells appear on the cornea; they are minute, less than the size of a pin's head, and are often arranged in rows or groups. They quickly desquamate, forming erosions which may heal rapidly, leaving no opacity but are accompanied by great irritation, lacrimation, and blepharospasm. Usually, however, fresh crops appear, and the con-

dition may prove very obstinate. After the plaques have desquamated, leaving minute shallow clear facets, the shape of the lesions and the total absence of vascularization are distinguishing features as well as their grouping, the crenated edges where several have coalesced, and the persisting filamentary shreds of diseased epithelium. In all cases the cornea is relatively insensitive.

In severe forms *dendritic ulcers* develop. The infiltrates, spreading in all directions, coalesce with each other and form a large shallow ulcer with crenated edges. More often grey striæ extend in one or more directions, increase in length, and send out lateral branches which are generally knobbed at the ends so that a dendritic figure, not unlike a liverwort, is formed which resembles no other condition and is pathognomonic (Fig. 168). The surface over the infiltrates

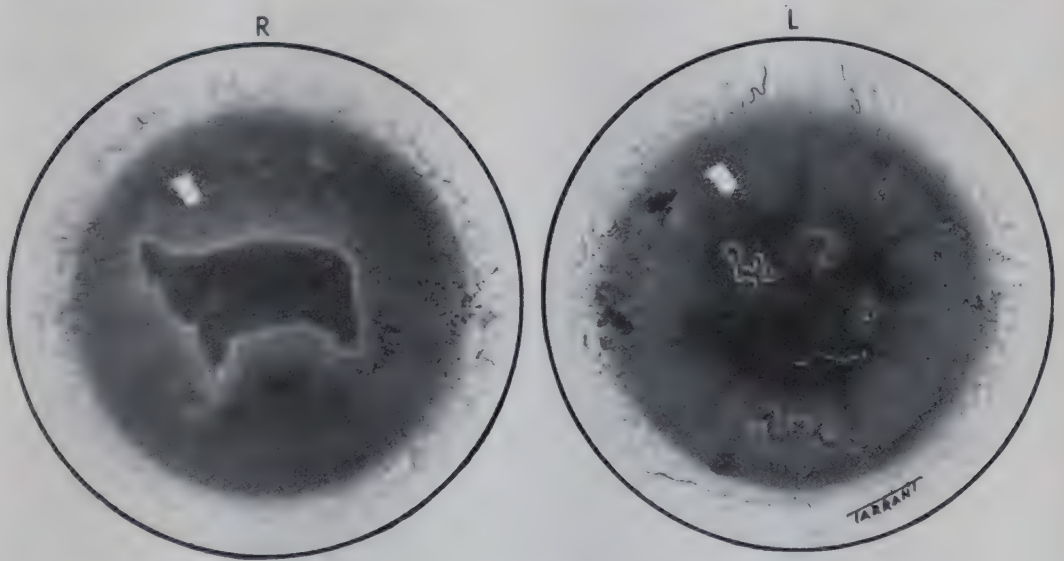


FIG. 168. Herpetic ulceration. In the right eye a large confluent ulcer with outlying areas of staining; in the left eye, multiple dendritic ulcers (Barrie Jones).

breaks down and an extremely irritating and chronic type of ulcer is produced, persisting with exacerbations for weeks or months, sending out fresh branches but never extending in depth. Generally only one or two of the infiltrates stain with fluorescein at any given time, but fresh spots are continually being formed and the disease not infrequently recurs; alternatively, a large confluent ulcer may be formed (Fig. 168). Meantime the stroma may be implicated and a *disciform keratitis* (*q.v.*), sometimes of considerable extent, may develop. An iritis is invariably associated with a severe herpetic keratitis; sometimes it is of considerable severity and occasionally a hypopyon occurs. The herpetic virus has been isolated from the aqueous in such cases.

Treatment. Specific treatment by the topical administration of antiviral substances is encouraging in early cases (p. 145). Apart

from this, atropine, warm compresses and bandaging are the most effective symptomatic measures. In intractable cases and in dendritic ulcers the most effective expedient is to destroy a wide area of epithelium by cauterization with carbolic acid or iodine (7 per cent. iodine and 5 per cent. potassium iodide in alcoholic solution) applied on a swab, following anæsthetization of the eye. This treatment has often to be repeated. After cauterization atropine is instilled and the eye bandaged. It is well to do this treatment at an early stage for there is evidence that it tends to prevent an extension of the disease deep into the corneal stroma to give rise to a disciform keratitis (*q.v.*). Corticosteroids which suppress the immunological reaction should never be given.

Healing may be aided by the administration of vitamin C (ascorbic acid) given at first intravenously on alternate days (500 mg., 4 to 6 injections) followed by 2 tablets of 250 mg. by mouth three times a day. It has been found that repeated vaccination (as for smallpox), given as a course once a week for 6 to 8 consecutive weeks, sometimes tends to prevent recurrences in herpes carriers who are periodically subject to attacks of keratitis.

Zoster. (Herpes) Zoster is caused by a virus identical with that causing chicken-pox; the two diseases may be associated in



FIG. 169. Left-sided zoster affecting the ophthalmic division of the Vth nerve with the naso-ciliary branch.

epidemics and it may be that after an infection with chicken-pox in youth the virus lies dormant to appear later, particularly in elderly people, causing the clinical picture of zoster. In zoster ophthalmicus the chief focus of infection is the gasserian ganglion whence the virus travels down one or more of the branches of the ophthalmic division of the fifth nerve, so that its area of distribution is marked out by rows of vesicles or the scars left by them, exactly as in zoster in other parts of the body (Fig. 169). The supra-orbital, supra- and infra-trochlear branches are nearly always involved ; frequently the nasal branch ; only rarely the infra-orbital branch. It is very rarely bilateral. There may be fever and malaise at the onset, and the eruption is preceded by severe neuralgic pains along the course of the nerves which are so characteristic of zoster that they should arouse

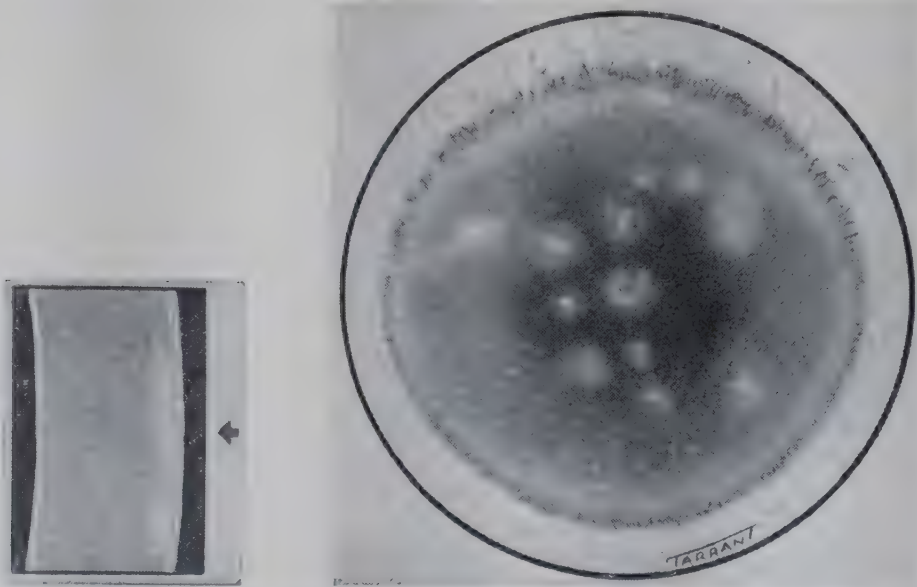


FIG. 170. Zoster of the cornea. The large maculæ are zoster lesions ; the small dots below are keratic precipitates.

suspicion of the nature of the disease before the vesicles appear. The pain sometimes ceases after the outbreak of the eruption, but sometimes it may continue for months or even years. The skin of the lids and other areas affected becomes red and œdematous, so that the disease may be mistaken for erysipelas, but the characteristic distribution and especially the strict limitation to one side of the middle line of the head should obviate this error. The vesicles often suppurate, bleed and cause small, permanent, pitted scars. The active eruptive stage lasts about three weeks and is followed by some anæsthesia of the skin. Ocular complications arise during the subsidence of the eruption, but may be overlooked during the acute stage owing to the difficulty in examining the eye ; they are generally associated with involvement of the naso-ciliary branch of the trigeminal (Fig. 170).

With the slit-lamp, numerous rounded spots composed of minute white dots are seen in the epithelium, which soon involve the stroma to form a coarse subepithelial punctate keratitis. Sometimes the infiltration spreads deeply and becomes diffuse in the stroma, associated with iridocyclitis (*q.v.*). The cornea is usually insensitive. This is tested by touching it with a wisp of cotton-wool, and comparing the reaction with that in the opposite eye; the slightest touch is followed by reflex closure of the lids if the cornea is sensitive. Similar nodules leaving grey scarred areas may appear on the sclera and patches of atrophy may develop on the iris. The intra-ocular tension is sometimes diminished in the early stage, but subsequently a secondary glaucoma is not unusual. The ocular lesions and the corneal anæsthesia are very obdurate and often persist long after the disease has otherwise passed away. In some cases there is associated paralysis of motor cranial nerves, especially the third, sixth and seventh, which usually passes off within six weeks. Facial palsy adds seriously to the danger to the eye owing to exposure of the cornea.

Treatment. Treatment is unsatisfactory and essentially symptomatic. The eye should be carefully examined in every case of zoster ophthalmicus, the lids being separated by retractors if necessary. Oily drops or methylcellulose and atropine should be instilled. It has been claimed that the injection of immune serum before the eye becomes involved may protect this organ; but this is questionable and such serum is not easy to obtain. Once ocular infection has developed keratitis and iridocyclitis must be treated in the usual manner. The skin may be treated with a sulphonamide powder or penicillin ointment, while in the early stages an ointment of hydrocortisone may arrest the development of the local lesions. No certain means are known to relieve the pain apart from rest and sedation.

Vaccinia affects the eye, usually as an accidental inoculation from a recently vaccinated child. The lids are usually affected (p. 488) but if the conjunctiva and cornea are involved an alarming clinical picture results, with swelling of the lids so intense that examination may be difficult. The pre- and post-auricular nodes are always involved. In the conjunctiva the typical lesion is extensive ulceration; in the cornea a punctate subepithelial keratitis which may progress to map-like ulceration and stromal abscesses. A disciform keratitis may develop at a later date. *Treatment* is by gamma-globulin from a recently vaccinated person used topically (250 mg. in 2.5 ml. distilled water every half-hour) and by intramuscular injections. Interferon used topically every half-hour may give spectacular results and IDU (p. 145) has been successfully employed.

Constitutional Affections

Phlyctenular Keratitis. It has already been pointed out that phlyctens are commonly found at the limbus; they may also occur

within the corneal margin. The fact must be emphasized that the disease is essentially conjunctival, and when the cornea is affected it is the conjunctival element of the cornea—the epithelium and the superficial layers underlying it—which suffers. As has already been noted it is an allergic reaction to an endogenous allergen, notably tuberculo-protein (p. 175).

Corneal phlyctens are localized infiltrations of exactly the same nature as conjunctival phlyctens but cause much pain and reflex blepharospasm (photophobia). They may become absorbed without destruction of the superficial layers of the stroma, in which case they cause no permanent opacity. The epithelium, however, is readily destroyed and the denuded surface easily becomes infected, usually by staphylococci (p. 186) ; in this manner a small superficial ulcer is formed. The corneal phlycten is a grey nodule, slightly raised above the surface and if the epithelium breaks down, a yellowish ulcer is formed. The subsequent course depends upon the nature of

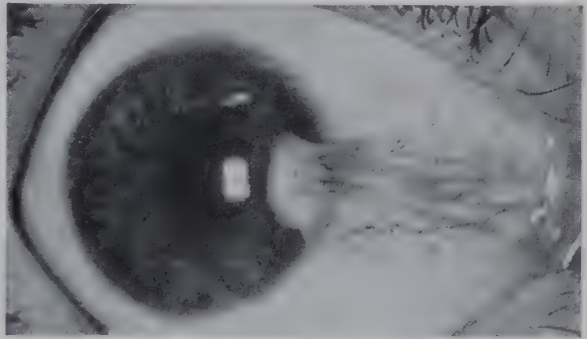


FIG. 171. Fascicular ulcer.

the infection and the nutritional state of the patient. The ulcer may deepen rapidly and even perforate, although this is uncommon.

A very characteristic form of phlyctenular ulcer is the *fascicular ulcer* (Fig. 171). This is a serpiginous ulcer which steadily creeps over the cornea, usually towards the centre, advancing slowly for weeks. It is supplied by a leash of vessels which lie in a shallow gutter and follow the advance of the ulcer. The ulcer starts near the limbus and heals on the peripheral side, while the central margin remains grey and infiltrated. As long as this infiltrated crescent is seen the ulcer is progressing, but it always remains superficial and never perforates. When healing finally takes place the vessels gradually disappear, but the whole of the track of the ulcer remains as a permanent opacity, densest, however, where the ulcer stops. In the severest cases a diffuse deep-lying infiltration forming a large greyish area may develop which may become yellow and break down, forming a large ulcer.

Sometimes the phlyctens are so closely packed at the limbus that they become confluent and may even surround the cornea. If they

break down and form a *ring ulcer* an extremely dangerous condition results. The nutrition of the whole cornea is endangered and even if total necrosis does not occur an extensive perforating ulcer may be formed at the margin. Such an outcome is fortunately rare.

More commonly the continuous infiltration of the limbus leads to the development of superficial vessels at the periphery of the cornea, a condition which is called *phlyctenular pannus* (pannus eczematosus or scrofulosus). Unlike trachomatous pannus (*q.v.*), it shows no special predilection for the upper part of the cornea. It is thin and not very vascular and usually clears to a large extent, although the course is generally very tedious. It is accompanied by intense blepharospasm.

Treatment of phlyctenular keratitis is the same as that of phlyctenular conjunctivitis (*q.v.*) until ulceration has occurred when atropine combined with corticosteroids should be intensively administered as drops or ointment, associated with vitamin C in large doses. If the ulcer still progresses despite these medicaments the crescentic infiltrate should then be cauterized with pure carbolic acid or the actual cautery, and the vessels may be destroyed with the cautery. If a fascicular ulcer has already reached the centre of the cornea and lies over the pupillary area it is not a bad plan to allow it to progress still farther, because the opacity left by the track of vessels is less dense than that left at the final site of ulceration.

In *acne rosacea*, seen generally in elderly women, keratitis associated with much irritability and lacrimation may occur. In addition to slight muco-purulent conjunctivitis, yellowish white infiltrates and small ulcers appear in the cornea which always becomes heavily vascularized. They are very intractable and frequently recur. In severe cases there is also iritis.

Local treatment is disappointing but should be similar to that for phlyctenular keratitis; the greatest relief usually follows the instillation of corticosteroids as drops or ointment. The essential treatment, however, is that of the skin condition. When the skin lesions are active, mild applications (1/3 of an erythema dose) of X-rays have been found to be beneficial. Constitutional treatment, however, may be necessary; unless this is undertaken with thoroughness and persistence recurrences are invariable and progressive corneal damage constant.

Ariboflavinosis. Deficiency of riboflavin (vitamin B₂ or G) in the diet leads to peripheral corneal vascularization, due to proliferation of capillaries from the normal sclero-corneal loops, and photophobia. The vessels are best seen with the slit-lamp, but their diagnostic importance has been exaggerated. Ariboflavinosis should not be diagnosed in the absence of other signs of deficiency. In the fully developed condition the lips are purplish and cracked at the angles (*cheilosis*); the tongue is inflamed (*glossitis*) and fissured, with enlarged

papillæ. There is seborrhœa of the naso-labial folds, eyelids and ears, and follicular keratosis of the forehead, malar eminences, and chin. Food rich in vitamin B₂ should be given.

Photophthalmia caused by ultra-violet rays, especially from 311 to 290 m μ , is characterized by extreme burning pain, lacrimation, photophobia, blepharospasm, and swelling of the palpebral conjunctiva and retrotarsal folds. It is due to desquamation of the epithelium to form multiple erosions. There is a latent period of four or five hours between the exposure and the onset of symptoms. The condition is generally caused by the bright flash of a short circuit or exposure to a naked arc light, as in industrial welding or cinema studios. It is rarely due to exposure to enclosed arc lights since the glass globe absorbs the most deleterious rays. In *snow blindness* the cause and symptoms are similar, for the ultra-violet rays are reflected from snow surfaces.

Prophylaxis consists in the wearing of dark glasses when such exposure is to be anticipated, particularly made of such materials as Crookes's glass which cuts off practically all the infra-red and ultra-violet rays. The *treatment* for both types of photophthalmia is by cold compresses and astringent lotions. Comfort will be obtained by bandaging both eyes for twenty-four hours until the epithelium has regenerated. Cocaine, which hinders epithelial healing, should be avoided, but atropine brings some relief.

Deep Forms of Keratitis

The deep forms of keratitis comprise that due to congenital syphilis, tuberculosis or virus infections (disciform keratitis) as well as lesions of indefinite origin or due to the spread of scleral inflammations (sclerosing keratitis, p. 223).

Interstitial keratitis (parenchymatous keratitis) is an inflammation affecting chiefly the stroma of the cornea. It is of infective or more often of allergic origin.

Interstitial Keratitis due to Inherited Syphilis affects most commonly children between the ages of five and fifteen. Delayed interstitial keratitis occasionally occurs in patients over thirty and is more liable to be unilateral but is often severe; the inflammation usually follows an injury to the eye, such as a blow or an operation, the injury acting as an exciting cause in a subject naturally prone to the disease, usually a congenital syphilitic. After slight irritative symptoms with some ciliary congestion, one or more hazy patches appear in deep layers of the cornea near the margin or towards the centre (Plate VI, Fig. 3). If they start near the margin they migrate towards the centre; if at the centre, others appear and fuse, until finally the whole cornea looks lustreless and dull. In two to four weeks the whole cornea is hazy with a steamy surface, giving a general appearance resembling ground glass in which denser spots can always be seen. As a rule the iris can be seen dimly, but in the severest cases the whole cornea becomes opaque, so that this tissue is hidden.

Meanwhile vascularization has occurred. It is of the deep type (Fig. 105, p. 103), consisting of radial bundles of brush-like vessels, and since they are covered by a layer of hazy cornea, their bright

scarlet is toned down to a dull reddish pink ("salmon patches") wherein separate vessels can be seen only with difficulty (Fig. 172). The opacity extends a little beyond the vessels, which seem to push the opacity in front of them and at the height of the condition the vessels run in radial bundles almost, but seldom quite, to the centre of the cornea. There is often a moderate degree of superficial vascularization, greater in some cases than in others, but never extending far over the cornea, and at the limbus the conjunctiva may be heaped up like an epaulette. In the absence of treatment this florid stage usually lasts for two to four months when subjective symptoms are very marked—pain, intense photophobia, lacrimation and blepharospasm are so severe that to open the eyes may be difficult or impossible. Vision may be reduced to the appreciation of hand-movements.

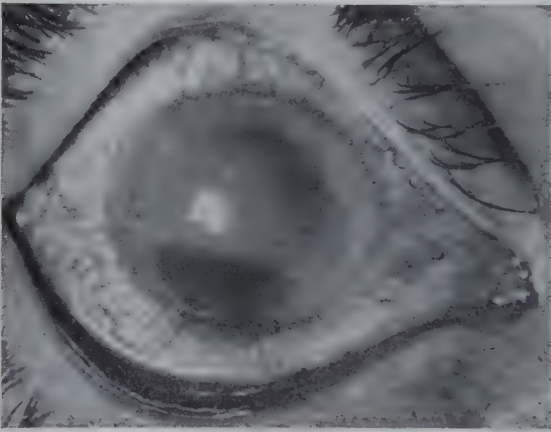


FIG. 172. Syphilitic interstitial keratitis with salmon patch below.

After the disease has reached its height the cornea clears slowly from the margin towards the centre, which may long remain hazy, but finally improves except in the worst cases. As the cloudiness disappears the vessels become obliterated, but although they cease to carry blood they remain permanently as fine opaque lines the characteristic radial course of which indicates the previous occurrence of the disease.

Since the infiltration of the cornea is almost entirely limited to the deeper layers lying immediately anterior to Descemet's membrane the corneal surface rarely becomes ulcerated. It is frequently stippled, steamy, and slightly uneven, and this condition may persist. In the worst cases the cornea may be enormously thickened and gelatinous in appearance giving the impression that it is ectatic and that the eye is in a hopeless condition, but it usually improves with some useful vision. In no case must an eye be removed on account of interstitial keratitis.

In interstitial keratitis the uveal tract is always profoundly affected and a considerable degree of iritis is invariably present. Sometimes there is severe cyclitis, as shown by the presence of

keratic precipitates on the back of the cornea and not infrequently a choroiditis, particularly around the periphery. Indeed, the disease is fundamentally a uveitis, and the keratitis, which clinically masks the uveitis, is secondary. It is important to realize the true pathology, since treatment must be directed to avoiding the deleterious results of iridocyclitis rather than those of keratitis.

Interstitial keratitis is almost invariably bilateral, although an interval of three or more weeks usually intervenes before the onset in the second eye ; rarely the interval is several months. The acute stage lasts at least six weeks and may extend to several months. The clearing of the cornea takes many weeks or months, but little improvement can be expected after eighteen months.

Diagnosis. A diagnosis depends on other evidences of congenital syphilis and positive serological reactions to this disease. It will be remembered that the most characteristic signs of congenital syphilis are prominence of the frontal eminences, flatness of the bridge of the nose, Hutchinson's teeth (notching of the two upper central incisors in the permanent dentition), rhagades at the angles of the mouth, shotty cervical nodes and periosteal nodules particularly on the tibiae.

Treatment. It is usual to order antisyphilitic remedies, but it is doubtful if they have any influence over the course of the keratitis, partly because the cornea is non-vascular and partly because the reaction is probably largely allergic. It is possible, however, that intensive systemic treatment with penicillin combined with malaria or other fever therapy (p. 147) may shorten the course of the disease.

Local treatment consists primarily in guarding against the evil effect of the uveitis which is an invariable accompaniment of the disease. Atropine is ordered as a routine measure, with the double purpose of keeping the ciliary body and iris at rest and preventing the formation of posterior synechiæ. Hot bathings or radiant heat should be used frequently in the acute stage. The most effective measure, however, is the topical administration of corticosteroids by drops or ointment (p. 145). With intensive treatment the response to this hormone may be dramatic in the early stages and in a few days the symptoms subside, the injection disappears and the eye becomes white. If the treatment is started at a very early stage the attack may thus be aborted, but if it is delayed until necrosis of the stroma has occurred, permanent opacities remain. Particularly in cases wherein the treatment has been instituted at an early stage, however, relapses are common as soon as the treatment is stopped ; these should therefore be anticipated and, indeed, it is wise in all cases to continue maintenance doses of steroids (as drops twice a day or as ointment at night) for at least a year until the natural period of the disease has elapsed.

In cases wherein pain and blepharospasm are severe, relief for a week or so may be obtained by the retro-ocular injection of 1.5 ml. of pro-

caine (4 per cent.) into the region of the ciliary ganglion, followed seven minutes later by 1 ml. of alcohol (40 per cent.). The pain and blepharospasm are relieved, vascular congestion much reduced, and the child is able to tolerate light.

In the later stages the means used for clearing corneal cicatrices may be tried. In severe bilateral cases, however, the best results are obtained by corneal grafting, which must be of the penetrating type. In this disease the prognosis for grafting is generally good.

Interstitial Keratitis due to Other Causes. In *acquired syphilis* interstitial keratitis is uncommon ; when it occurs it is generally unilateral. Several cases have been reported in which the primary lesion has been on the lids or face, and the keratitis has been limited to the same side. Most of the reported cases have occurred about two years after the primary sore, but the interval may be much shorter or longer. The clinical characteristics are similar to those of the congenital variety.

In *tuberculosis* a somewhat similar picture occurs. Thus, in tuberculosis of the iris (*q.v.*) it is not uncommon in the late stages to meet with

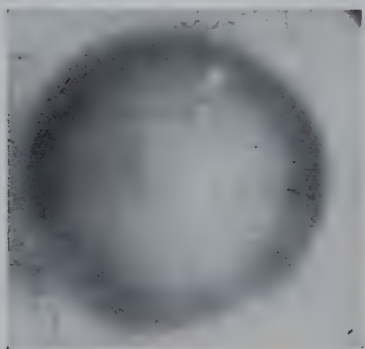


FIG. 173. Tuberculous interstitial keratitis.

an interstitial keratitis. Similar cases appear in the absence of ocular tuberculosis ; frequently these show a marked positive skin reaction to tuberculo-protein (Fig. 173).

Interstitial keratitis also occurs, although rarely, in mumps, trypanosomiasis, brucellosis, malaria and other conditions.

Keratitis disciformis is not uncommon : it occurs generally in adults and is unilateral. It is probably, in most cases, associated with a virus infection and has long been known to result from vaccinia affecting the lid margins, or from herpes, and not infrequently develops from a superficial punctate keratitis. The condition is probably not due to a direct infection of the corneal stroma by the virus from the epithelium but seems to be the expression of a tissue-response, sometimes involving necrosis, due to the reaction between antigens liberated by the virus in the epithelium and antibodies produced in the stroma or carried thither by the bloodstream. The pathology may thus be analogous to that of syphilitic interstitial keratitis. The importance of early destruction of the infected epithelium in herpetic cases is thus obvious.

The disease is characterized by the gradual appearance of a

central grey disc lying in the middle layers of the stroma usually with a denser central opacity. The slit-lamp shows thickening of the cornea and often folds of Descemet's membrane. It is accompanied by moderate irritation which, however, persists for weeks or many months, leaving a permanent opacity. The cornea may become anæsthetic but ulceration does not occur. Owing to the central situation vision is considerably impaired. It is little amenable to treatment, but the symptoms and probably the depth of the permanent opacity may be ameliorated by the local administration of corticosteroids. In the worst cases, particularly when the cornea is anæsthetic, tarsorrhaphy may be indicated (*q.v.*).

DEGENERATIVE CHANGES IN THE CORNEA

A variety of degenerative conditions occurs in the cornea, many of which are of serious clinical import. These are conveniently divided into three categories: primary degenerations, secondary degenerations depending on long-standing changes in the eye itself, and infiltrations associated with metabolic disturbances.

Arcus senilis is a lipid infiltration of the cornea met with in old people. It commences as a crescentic grey line concentric with the upper and lower margins of the cornea, the extremities of which finally meet so that an opaque line, thicker above and below, is formed completely round the cornea. It is characterized by being separated from the margin by a narrow line of comparatively clear cornea, being sharply defined on the peripheral side, fading off on the central. It is never more than about 1 mm. broad and is of no importance either from the point of view of vision or of the vitality of the cornea (Fig. 174).

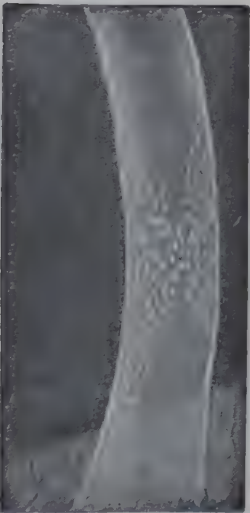


FIG. 174. Arcus senilis seen in corneal section with the slit-lamp.

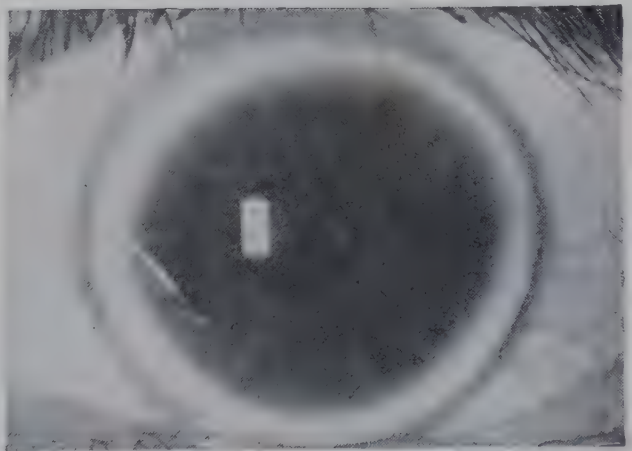


FIG. 175. Arcus juvenilis.

Arcus juvenilis is exactly similar to **arcus senilis** but is a rare condition found in children (Fig. 175). Even **arcus senilis** may develop at a comparatively early age, but the juvenile condition is probably congenital. It is of no importance. The characteristic diagnostic feature of both these opacities is the presence of a line of clear cornea between them and the limbus. This is occasionally found in old sclerosing keratitis, but in this case the opacity is usually localized to one part of the cornea and extends farther towards the centre.

Hereditary corneal dystrophies occur mainly in males about puberty as bilateral affections of obscure origin involving the central area of the cornea; they rarely affect the corneal margin. The lesions are characterized by the development of discrete areas of opacification mainly in the superficial layers of the stroma due essentially to hyaline deposits between the corneal lamellæ. They tend to increase in number and density until Bowman's membrane becomes eroded and the epithelium desquamates. Relatively symptomless and without inflammatory reaction, they progress slowly until the vision becomes seriously impaired usually over the age of 40. When this occurs treatment is best by a lamellar keratoplasty.

Three main forms have been differentiated. In the *nodular corneal dystrophy of Groenouw*, which has a dominant heredity, the opacities assume a granular form and subsequently coalesce into various irregular shapes (Fig. 176). A *lattice form*, also with a dominant heredity, is characterized by bifurcating criss-cross lines associated with punctate opacities. A *macular form* has a recessive heredity and in it the visual acuity tends to be affected at an early age.



FIG. 176. Nodular corneal dystrophy.

Endothelial corneal dystrophies occur rarely, the most common of which is the *endothelial dystrophy of Fuchs*. This is seen in elderly people, particularly females, commencing with fine changes in the endothelium distinguishable by the slit-lamp; these are followed by the formation of hyaline nodules on Descemet's membrane and the eventual atrophy of the underlying endothelial cells

(*cornea guttata*). These deep changes are followed by a diffuse dystrophy of the epithelium associated with œdema and the formation of vesicles and a grey opacification as well as punctate opacities in the stroma. Ultimately the entire cornea turns opaque and insensitive. Fortunately the course of the disease is slow so that blindness is usually forestalled by death. Treatment is difficult; corneal grafting is sometimes ineffective.

Senile marginal degeneration is also a rarity occurring in one or both eyes of old people. Bowman's membrane and the stroma suffer fibrillar degeneration and are eventually replaced by vascularized granular tissue. In the process a gutter forms slowly in the periphery of the cornea, starting

in the situation of an arcus senilis and eventually becomes ectatic (Fig. 177). Treatment is disappointing but reinforcement by a conjunctival flap or a lamellar keratoplasty may be tried.

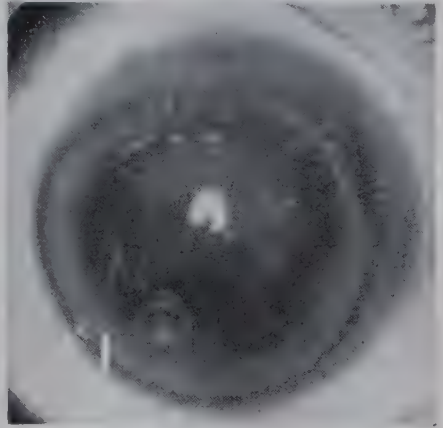


FIG. 177. Marginal degeneration of the cornea.

Band-shaped Keratopathy. This is a common condition in old, blind, shrunken eyes due to defective nutrition and exposure. It lies in the interpalpebral area, commencing at the inner and outer sides and progressing until it forms a continuous band across the cornea (Fig. 178). Close to the limbus, however, the cornea is generally relatively clear, as in so many degenerative conditions—probably owing to the better nutrition close to the blood-vessels. The condition is due to hyaline infiltration of the superficial parts of the stroma, followed by the deposition of calcareous salts.

It is rarely found, usually bilaterally, in otherwise healthy eyes as a horizontally oval area in the palpebral fissure.

Treatment. In the rare form last mentioned, improvement of vision may be obtained by scraping off the opacity, which is usually calcareous and quite superficial, or dissolving it with the sodium salt of ethylene-diamine tetra-acetic acid (p. 560). In the common

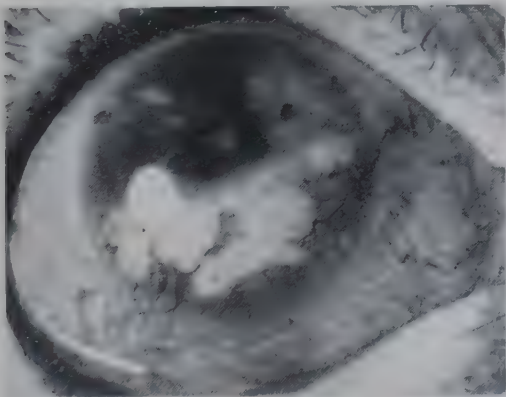


FIG. 178. Band-shaped keratopathy.
DIS. OF EYE.

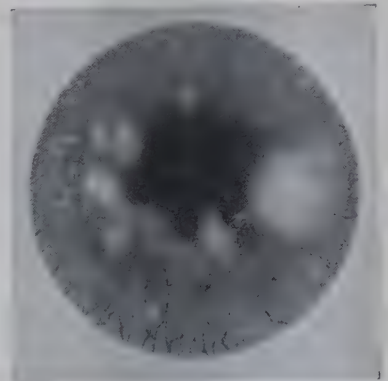


FIG. 179. Salzmann's corneal dystrophy.

form the eye is usually blind, and nothing remains but to remove it if it is painful or unsightly.

Other degenerative changes are frequently met with in old leucomata or anterior staphylomata consisting of hyaline infiltration and calcification. Such scars are liable to undergo a serious form of ulceration (p. 201).

A distinctive form is known as *Salzmann's nodular dystrophy*, a degenerative condition characterized by bluish-white nodules appearing in the superficial layers of the stroma and Bowman's membrane, occurring in persons who have suffered previous corneal disease (Fig. 179). The condition tends to be slowly progressive and may be treated by lamellar keratoplasty.

Infiltrations of the cornea are rare. A primary *lipid infiltration* of obscure origin may occur; it is a characteristic of gargoylism. Equally rare are primary *calcareous degeneration* and *dystrophia urica* wherein urate crystals form yellow opacities in the cornea. Similarly, deposits of *cystine* may be associated with a generalized cystinosis, renal dwarfism and osteoporosis (*Fanconi's syndrome*).

ECTATIC CONDITIONS

It has already been stated that ectatic conditions of the cornea may result from inflammation, as in keratectasia (p. 189) and anterior staphyloma (p. 191). Two forms of ectasia of non-inflammatory origin are known—keratoconus and keratoglobus.

Keratoconus (*Conical cornea*) is frequently due to a congenital weakness of the cornea, though often it only manifests itself after puberty, generally in girls. The cornea is thin near the centre and

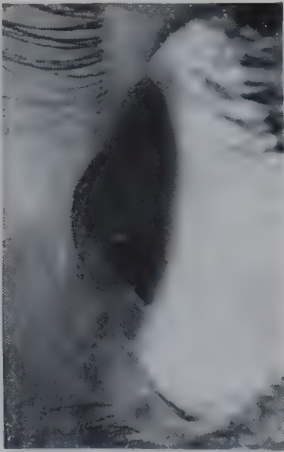


FIG. 180. Keratoconus.

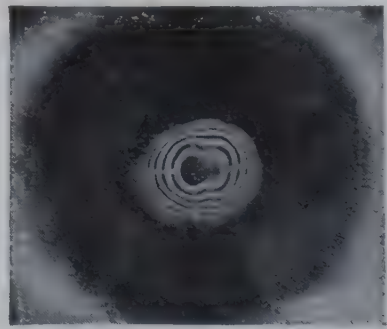


FIG. 181. Keratoscopic appearance of the corneal reflex in an advanced case of keratoconus.

progressively bulges forwards, the apex of the cone being always slightly below the centre of the cornea (Fig. 180). Sometimes it pulsates synchronously with the arterial pulse, and this may cause a subjective apparent pulsation of external objects. The cornea is

at first transparent, and vision is impaired through the protrusion and alteration in curvature. If the condition is marked the conical shape is easily recognized in profile, particularly by the acute bulge given to the lower lid when the patient looks down. In the less advanced cases distortion of the corneal reflex over the centre is the chief guide, a change best seen with Placido's disc or keratoscope (Fig. 181 ; p. 102) or when the cornea is examined with the ophthalmometer. With the ophthalmoscope mirror at a distance of 1 metre a ring of shadow concentric with the margin is seen in the red reflex, altering its position on moving the mirror. It is due to a zone through which few rays pass into the observer's eye owing to the fact that the emergent rays on the central side are convergent whilst those on the peripheral side are divergent.

The patient becomes myopic, but the error of refraction cannot be satisfactorily corrected with ordinary glasses owing to the parabolic nature of the curvature. The condition is almost invariably bilateral, though frequently more advanced on one side than the other. It may be slight and very slowly progressive, or the reverse. In the later stages the apex shows fine more or less parallel striæ, anastomosing at acute angles, best seen with the slit-lamp, and also discrete opacities which become confluent. A brownish ring, probably due to hæmosiderin, may form in the epithelium encircling the cone (Fleischer's ring). Sometimes ruptures develop in Descemet's membrane in which case the stroma becomes suddenly oedematous and opaque.

Treatment. In the early stage vision may be improved with spectacles, but contact lenses (p. 81) are more beneficial; in this condition they are usually well borne, they eliminate the irregular corneal curvature, and are said to have a supporting effect. If, however, the disease progresses two operative measures may be tried. The most satisfactory treatment in such cases is the removal of a large whole-thickness disc from the central area of the cornea and its replacement by a corneal graft. Corneal transplants are particularly successful in this condition and should be considered in progressive cases and whenever visual loss has become considerable. The classical treatment is deep cauterization of the apex with the actual cautery ; perforation here has been advocated, a procedure, however, not without danger to the eye from the formation of a corneal fistula, anterior synechia or infection. The scarring from cauterization is much less than might be anticipated, but a subsequent optical iridectomy may be advisable.

Keratoglobus is a hemispherical protrusion of the whole cornea, occurring bilaterally as a congenital anomaly: it is familial and hereditary. It differs from buphthalmos (*q.v.*) in that the intra-ocular pressure is normal, the cornea clear, the angle of the anterior chamber normal, and there is no cupping of the optic disc.

SYMPTOMATIC CONDITIONS

There are many pathological conditions of the cornea which are merely evidence of disease in other parts of the eye or of extension of disease. Some are often described as true diseases of the cornea, notably as forms of "keratitis"; this involves a wrong principle and a misuse of terms which can only lead to confusion. Since it is of great importance to distinguish these conditions from primary affections of the cornea from the points of view of both diagnosis and treatment, it will be well to review the more common here.

Corneal œdema is a relatively common condition. It manifests itself first in the epithelium which becomes steamy, an appearance due to the accumulation of fluid between the cells, especially the basal cells. At the same time the accumulation of fluid between the lamellæ and around the nerve-fibres of the stroma produces a haziness throughout the entire cornea due to alterations in the refractive condition. If the œdema lasts for a long period the epithelium tends to be raised into large vesicles or bullæ (*vesicular or bullous keratopathy*) (Fig. 182). This is a peculiarly intractable condition which frequently gives rise to intensely irritating symptoms as the bullæ periodically burst.

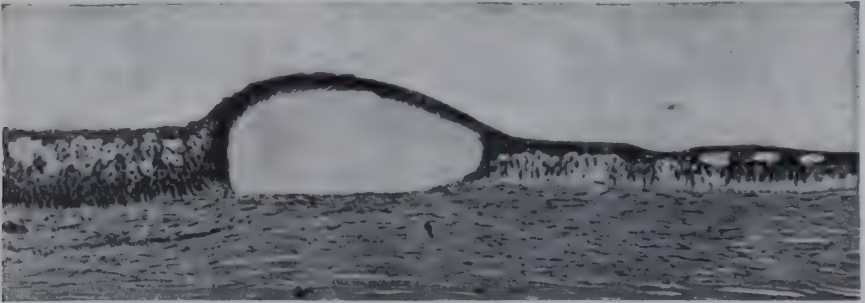


FIG. 182. Corneal œdema with a large vesicle (bullous keratopathy) in a case of long-standing glaucoma.

Such a corneal œdema may occur in many conditions of an inflammatory or degenerative type of long standing. It is common in long-standing glaucoma when the tension is high; it also tends to occur whenever the endothelium has suffered damage so that the aqueous can percolate through the stroma. After trauma or operation such an œdema is characteristic of endothelial damage, particularly when strands of vitreous remain adherent to the posterior surface of the cornea. Treatment is difficult unless the primary cause of the œdema can be eliminated. Occasionally—but only occasionally—some comfort may be obtained by the frequent instillation of a concentrated saline solution or an ointment containing 15 per cent. sodium chloride. An alternative which is frequently effective is to strip off the entire epithelium and to replace it by a complete flap of the conjunctival epithelium only.

Filamentary keratopathy. Closely allied to bullous keratopathy is the formation of epithelial threads which adhere to the cornea by one end while the other, which is often club-shaped, moves about freely. Such filaments occur in degenerative conditions or in long-standing corneal œdema; they may be seen in cases of viral keratitis, particularly of the herpetic type, in the collagenous diseases and in keratitis sicca (p. 508).

Keratic precipitates, initially badly termed "keratitis punctata" or "k.p.", are depositions of leucocytes and other cells on the back of the cornea in cyclitis and iridocyclitis and occasionally in choroiditis. The greatest care must be taken not to overlook them, since they may be almost the only objective sign of serious disease. They may be on the back of a clear cornea or the deeper layers may be infiltrated as a result of the intra-ocular inflammation. Their appearance and nature will be described in discussing their cause (pp. 230, 234).

Opacities of the cornea are usually secondary to inflammation but some other types may occur.

Congenital opacities of various kinds are sometimes encountered as developmental anomalies; others are due to injury received at birth. The latter are often temporary and diffuse due to œdema caused by ruptures of Descemet's membrane.

Striate opacity occurs in various forms. The commonest form is that seen after operations upon the globe in which a peripheral corneal section has been made, as in cataract extraction. Here, delicate grey lines run from the wound and may pass completely across the cornea; they disappear spontaneously as the wound heals and are due to slight folding of the cornea whereby Descemet's membrane and the adjacent lamellæ become wrinkled. Radial striæ are seen around wounds or ulcers; they are partly referable to the same cause, partly to distension of the interlamellar spaces by œdema. The fine hatching which is seen around ulcers and sometimes after tight bandaging is to be referred to similar causes.

White Ring Opacities (Coats). Occasionally rings or oval formations composed of very dense white spots, about 0.5 mm. in diameter, occur beneath Bowman's membrane. The cause is disputed but they frequently follow minor trauma such as results from the impaction of foreign bodies; they do not interfere with vision.

Pigmentation of the cornea may occur from the prolonged topical use of silver nitrate (*argyrosis*, p. 182). As in the conjunctiva it is due to the permanent impregnation of the elastic fibres and particularly Descemet's membrane with metallic silver.

A similar deposit of copper forms a pigmented ring of a grey-green or golden-brown colour round the periphery of the cornea in the region of Descemet's membrane and the deeper layers of the stroma when a copper foreign body is retained within the eye (*chalcosis* (p. 394)) and in hepato-lenticular degeneration (Wilson's disease) (the *Kayser-Fleischer ring*).

Blood in the cornea is rare. It may occur as a bright red spot or streak superficially at the margin, or as a greenish or rusty stain throughout the whole tissue. In the latter case it is derived from blood in the anterior chamber, usually associated with high tension—a relatively infrequent complication following contusions (p. 376).

Tumours of the cornea have been considered with those of the conjunctiva.

CHAPTER 17

DISEASES OF THE SCLERA

THE avascularity of the sclera and the lack of reaction of its dense fibrous tissues to insult whether traumatic or infective, make diseases of this tissue relatively rare ; for the same reason, when they do occur they tend to be chronic and sluggish.

INFLAMMATION OF THE SCLERA

Two forms of inflammation of the sclera are described : superficial or episcleritis, and deep or scleritis. They might equally well be considered mild and severe forms of the same disease, but the distinction is convenient since they differ in their evolution.

Episcleritis is an inflammatory affection of the deep subconjunctival connective tissues, including the superficial scleral lamellæ and frequently affecting both eyes. A circumscribed nodule of dense leucocytic infiltration, which may be as large as a lentil, appears usually two or three millimetres from the limbus (Plate VII, Fig. 1). It is hard, immovable and tender, the conjunctiva moving freely over it, and is traversed by the deeper episcleral vessels so that it looks purple, not bright red. It is usually transient lasting several days or some weeks but has a strong tendency to recur ; in this manner the disease may drag on for months. Occasionally the attacks are fleeting but frequently repeated (*episcleritis periodica fugax*). On the other hand, the disease may be extremely chronic, in which case it never ulcerates and may be entirely absorbed, but more frequently leaves a slate-coloured scar behind to which the conjunctiva is adherent. The cornea and uveal tract rarely participate in the inflammation. There may be little or no pain, but usually there is a feeling of discomfort and tenderness on pressure, and often severe neuralgia. In the worst cases the disease extends into the deeper parts of the sclera and thus passes almost imperceptibly into scleritis.

Anatomically dense lymphocytic infiltration of the subconjunctival and episcleral tissues is found.

Rheumatism and gout were commonly indicated as the chief causes of episcleritis, but it is now more frequently regarded as an allergic reaction to an endogenous toxin (tuberculous, streptococcal, etc.) ; a history of acute rheumatism is rarely obtained. Alternatively, the condition is sometimes considered to lie within the vague category of collagenous diseases. It is commonest in women.

Treatment is difficult and often unrewarding. General measures are of more value than local, although warm compresses often give great relief. Corticosteroids administered topically as drops or ointment are sometimes of temporary benefit. General treatment should be directed in the first place to the elimination of focal infection and the desensitization of the patient (as by tuberculin, a streptococcal vaccine, etc.). Even in cases in which no history of rheumatism can be elicited, salicylic preparations seem to do good, and should be tried.

Scleritis is usually a bilateral disease, occurring most frequently in women, more rare than episcleritis and due to the same causes. One or more nodules may appear but the area affected is less circumscribed than in episcleritis. The swelling is at first dark red or bluish, later it becomes purple and semi-transparent like porcelain. It may extend entirely round the cornea, forming a very serious condition known as *annular scleritis*. In the diffuse type hard whitish nodules, the size of a pin's head, may develop in the inflamed zone ; they disappear without disintegrating. Scleritis differs from episcleritis in that the cornea and uveal tract are involved, some iritis but more usually cyclitis and anterior choroiditis being present. There is no ulceration, but much absorption so that the sclera is thinned, a dark purple cicatrix being formed which occasionally becomes too weak to withstand the intra-ocular pressure, so that ectasia follows (*ciliary staphyloma*). Secondary glaucoma is a common sequel (Plate VII, Figs. 2 and 4).

Pathologically scleritis resembles episcleritis, but extends more deeply, the essential change being a dense lymphocytic infiltration.

Apart from the intra-ocular complications scleritis may sometimes extend to the cornea causing *sclerosing keratitis* (Plate VII, Fig. 3). An opacity develops at the margin of the cornea near the affected scleral area ; it is approximately triangular or tongue-shaped, the rounded apex being towards the centre of the cornea. Similar opacities may develop farther from the margin and even at the centre. These are grey or greyish yellow, becoming denser until they exactly resemble the sclera—hence the name. They are due to changes in the stroma, which is embryologically a specially differentiated part of the sclera. There is little or no corneal vascularization and ulceration never occurs. Some clearing occurs from the centre towards the periphery as well as near the corneal limbus, but the densest parts usually persist as bluish clouds. The whole margin of the cornea may become opaque like the sclera, but the pupillary area almost invariably escapes.

Treatment is the same as for episcleritis but the uveal complications must be treated.

Gumma of the Sclera is uncommon. It may be indistinguishable in appearance from scleritis, or it may take the form of nodules of

various sizes situated near the limbus, extending backwards to the equator or even giving rise to an annular scleritis. A gumma may spread to the interior of the eye or a gumma of the ciliary body may spread outwards and involve the sclera. Unless active antisyphilitic treatment is adopted early and carried out thoroughly, the eye may be lost from uveal complications, ciliary staphyloma, or phthisis bulbi. The diagnosis depends upon the history, the co-existing signs of syphilis, and the application of serological tests. General antisyphilitic treatment is indicated.

Tuberculosis of the Sclera may take the form of a scleritis, may be an extension from the conjunctiva, iris, ciliary body, or choroid, or may be primary, forming a localized nodule which caseates and ulcerates. It should be excised or scraped and the tissue examined for the organism. Treatment consists of the local and systemic use of streptomycin together with the systemic administration of PAS or Isoniazid.

In the **Collagen Diseases** scleral implication is common, particularly in rheumatoid arthritis. Histologically the typical lesion assumes the

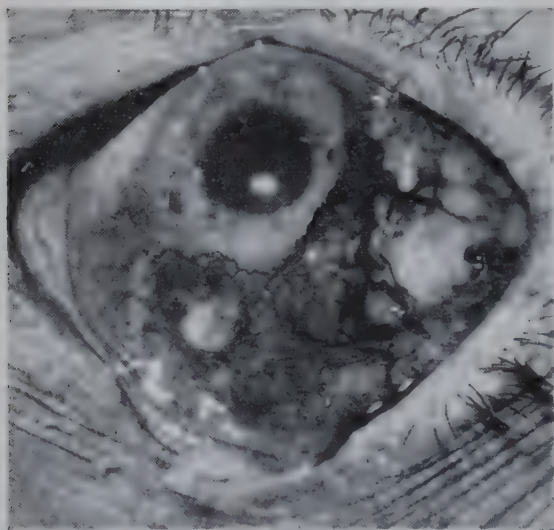


FIG. 183. Necrotizing nodular scleritis.

characteristic combination of a proliferative infiltration by chronic inflammatory cells surrounding a central area of fibrinoid necrosis, as occurs in rheumatoid nodules generally. In the sclera the clinical manifestations take several forms. *Episcleral rheumatoid nodules* may appear and disappear, waxing and waning with the vagaries of the systemic disease. A more serious condition is *necrotizing nodular scleritis* wherein a violent and painful anterior scleritis, often circumferential in its extent, characterized by much swelling and the appearance of one or more yellow nodules, usually proceeds to necrosis leading eventually to disintegration of the sclera and exposure of the underlying uvea (Fig. 183). In *scleromalacia perforans* the same necrosis of the sclera occurs with exposure of the uvea but without painful symptoms (Fig. 184). Finally, in *massive granuloma of the sclera*, proliferative changes are predominant. In the anterior segment of the eye a "brawny scleritis" results; in the posterior segment the sclera may become so

thickened as to simulate an intra-ocular or orbital tumour. In all these cases serious and often destructive extension occurs into the uveal tract, and the general prognosis is poor. Local and systemic treatment by corticosteroids affords the only known method of amelioration.

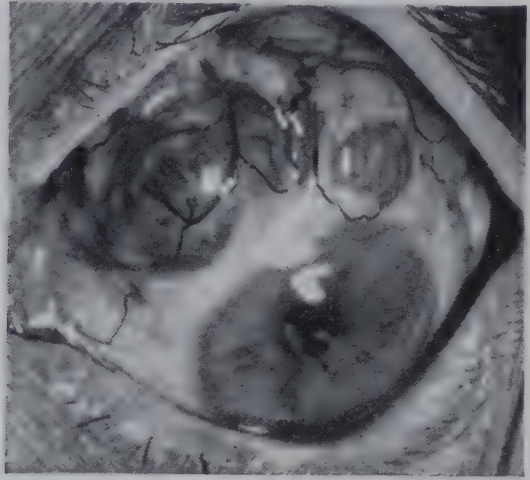


FIG. 184. Scleromalacia perforans.

Blue Sclerotics. The sclera is bluish in babies, but a much more pronounced blue coloration is sometimes seen in several members of the same family as a hereditary condition which persists throughout life ; it is frequently associated with fragilitas ossium and deafness. The sexes are about equally involved ; only those affected can transmit the disease. Histologically the sclera and cornea are thin so that the uveal pigment shines through to produce the blue colour.

CHAPTER 18

DISEASES OF THE UVEAL TRACT

ALTHOUGH topographically apparently separate, the iris, the ciliary body and the choroid are so closely related as to form a continuous whole; the diseases affecting the whole uveal tract will therefore be considered together.

VASCULAR DISTURBANCES

While the blood supply of the uveal tract is almost entirely derived from the posterior ciliary arteries, the peculiar distribution resulting in the formation of the major arterial circle of the iris causes involvement of both the iris and the ciliary body in pathological vascular conditions, whereas the blood supply to the choroid, being essentially segmental, results in the formation of lesions restricted to isolated areas. The richness of anastomoses in the anterior part of the uveal tract precludes isolated vascular lesions, but sclerotic changes in the choriocapillaris may be sharply delimited as occurs, for example, in macular degeneration. Localized choroidal hæmorrhages occur, as for example, in myopic degeneration, but these are difficult to diagnose from the rounded hæmorrhages which occur deeply in the retina. Massive hæmorrhages in the choroid occur in expulsive hæmorrhage.

Rubeosis Iridis is a curious and serious condition which occurs in diabetes of long standing and in thrombosis of the retinal veins. It is characterized by the development of new and enlarged vessels in the iris, the neo-vascularization being frequently accentuated towards its root and in the angle of the anterior chamber. It may be associated with signs of iritis, and a rise in tension often occurs leading to the development of a very intractable secondary glaucoma. Treatment is often difficult; an iridencleisis or partial destruction of the ciliary body by diathermy or cryosurgery may give relief.

INFLAMMATIONS OF THE UVEAL TRACT

The term *uveitis* emphasizes the close relationship between the anatomically distinct parts of the uveal tract, for inflammatory processes tend to involve the uvea as a whole and are not limited to a single part. This feature is particularly well exemplified in inflammation of the iris and ciliary body; iritis never occurs without some cyclitis, nor cyclitis without some iritis and the terms *iritis* and *cyclitis* are used according to whether the iris or ciliary body appears clinically to be the more affected. The same disposition is also seen with regard to the choroid, although in less degree, for

the ciliary body is often involved in many cases of choroiditis. A more correct terminology is *anterior* and *posterior uveitis*, while *general uveitis* is commonest in chronic types of inflammation.

The Ætiology of Uveitis

The determination of the ætiology of inflammations of the uveal tract is one of the most difficult problems in ophthalmology ; indeed, so great is our ignorance in this respect that in most cases of iridocyclitis or choroiditis we are in the dark as to the nature of the causal mechanism and must rely on guesses which have little logical basis. In some cases, it is true, the ætiology is obvious and several infections have distinguishing clinical features ; but in most cases which present a nondescript clinical picture, our ignorance is profound. It would seem probable that most of these are not due to direct infection but are allergic or constitutional in origin.

The following classification may prove useful :

(1) *Exogenous Infections*. These are rare and are due to the introduction into the eye of organisms through a perforating wound or ulcer. This results usually in an acute iridocyclitis often of a suppurative type, and sometimes in a panophthalmitis wherein the whole interior of the eye is involved.

(2) *Secondary infections*, wherein the inflammation of the uveal tract is due to spread from one or other of the ocular tissues—the cornea, sclera, or retina.

(3) *Endogenous infections*, wherein organisms primarily lodged in some other organ of the body reach the eye through the blood-stream. These comprise bacterial infections as tuberculosis, syphilis, gonorrhœa, or brucellosis, viral infections as in mumps, smallpox or influenza wherein an iridocyclitis occurs, and protozoal infections such as toxoplasmosis. The same mechanism causes the violent panophthalmitis seen in septicæmia due to such organisms as the streptococcus, staphylococcus, meningococcus or pneumococcus : in these the inflammation is suppurative in type. More doubt, however, exists as to the role of the subacute intermittent types of bacteræmia said to be associated with focal infections due principally to the streptococcus. It is probable that the mechanism of uveitis in the majority of such cases is allergic rather than infective.

(4) *Allergic inflammations* are generally considered to be common. Therein a primary source of infection exists somewhere in the body ; at one time the infection was generalized by the escape of organisms into the blood-stream when the ocular tissues had become sensitized to them ; and at a later date a renewal of activity in the original focus leads to a further dissemination of the organisms or their proteins which, meeting the sensitized ocular tissues, excite therein an allergic response. The two most favoured sources for this reaction are a tuberculous infection—usually a minute focus in the lung or a lymph node contracted initially in youth, apparently healed and

difficult to determine clinically—and the streptococcal infections which commonly abound in such sites as the teeth, sinuses, tonsils or prostate. It is significant that this condition is not associated with active pulmonary lesions in the first case, nor with a gross suppurative infection in the second, but rather with a mild and chronic focus which has been present for many years so that tissue-sensitivity has had time to develop. The type of uveitis generally accepted as being thus excited is the typical exudative variety, and many authorities ascribe the majority of cases of iridocyclitis to this cause; others view this mechanism with scepticism. In many cases the presence of sensitivity can be demonstrated by skin-reactions to tuberculo-protein or streptococci, and although the mechanism probably does account for the incidence of a large number of cases of plastic iridocyclitis of moderate severity, especially those which are recurrent or chronic in nature and are often associated with vague manifestations of muscular rheumatism ("rheumatic iritis"), its importance cannot yet be accepted as definitely assessed.

(5) *Constitutional disorders* undoubtedly determine the incidence of some cases of inflammation of the uveal tract, the most common being diabetes, gout and the collagenous diseases. In most cases, however, it is probable that the constitutional disturbance is not the actual cause but rather lowers resistance so that other ætiological factors can become more easily effective.

The relation between uveal inflammation and diseases of the joints is close; uveitis frequently occurs with rheumatoid diseases both in children (Still's disease) and adults; the two are associated in several syndromes (Behçet's, Reiter's, etc.), and one of the commonest concomitants of *anterior uveitis*, particularly in males, is *spondylitis*. It would seem, indeed, that the latter is frequently linked with a non-specific urethritis and prostatitis or a cervicitis, whether due to a virus, pleuropneumonia-like organism, or other cause. It is thus probable that many cases of anterior uveitis share a common ætiology with articular disease, sometimes associated with uro-genital infection (p. 549).

The General Course of (Non-purulent) Anterior Uveitis

Iritis. In order that iritis and the special dangers which attend it may be thoroughly understood, it is necessary to remember the anatomical arrangements of the iris and the pathological changes which occur in it. The iris is practically a diaphragm of blood vessels and unstriped muscle fibres held together by a loose, spongy stroma. In its perpetual movements the pupillary margin slides to and fro upon the lens capsule. The more the pupil is constricted the more of the posterior surface of the iris is in contact with the lens capsule; when fully dilated the iris may not touch the lens.

Inflammation of the iris has fundamentally the same characteris-

tics as in other connective tissues : dilatation of the blood vessels occurs with impairment of the capillary walls and exudation of a protein-rich fluid into the tissue spaces with leuco- or lymphocytosis. Owing to the extreme vascularity of the iris, the peculiar distribution of the vessels and the looseness of the stroma, these generic features of inflammation produce special results. Thus hyperæmia tends to cause the pupil to contract mechanically on account of the radial disposition of the vessels. The extreme vascularity and the looseness of the tissues allow an unusually large amount of exudation on the one hand and swelling on the other. The iris virtually becomes a water-logged sponge full of sticky fluid so that its freedom of movement is impaired, and the normal pupillary reactions become sluggish or abolished. The fluid also contains toxic substances which act as irritants causing the muscle fibres to contract, and since the sphincter overcomes the dilatator muscle, constriction of the pupil results. It follows that in iritis the pupil is constricted, its reactions are sluggish, and the delicate pattern of the iris instead of being clear and sharply defined, becomes blurred and indistinct ("muddy" iris). The colour undergoes considerable change ; in fair people the blue iris becomes bluish or yellowish green ; brown irides show less difference, but become greyish or yellowish brown. In any case, comparison of the colour of the two irides will usually reveal some difference, for iritis is generally unilateral during the acute attack.

The hyperæmia manifests itself in circumcorneal ciliary congestion, most marked if the ciliary body is seriously involved (p. 100). The conjunctival vessels are also frequently engorged so that care is necessary in distinguishing the condition from conjunctivitis, but the secondary nature of the conjunctival congestion is shown by the relatively slight discharge : any discharge is lacrimal, never mucopurulent.

Since the iris is richly supplied with sensory nerves from the ophthalmic division of the fifth nerve, pain, typically worst at night, is a prominent symptom of acute iritis. It is not confined to the eye, although severe neuralgic pain is felt here, but is also referred to other branches of the nerve, especially to the forehead and scalp, to the cheeks and malar bone, and sometimes to the nose and teeth.

The albuminous exudates escape into the anterior chamber and, particularly if the ciliary body is involved, the aqueous becomes plasmoid (p. 15) containing leucocytes and minute flakes of coagulated protein, or even fibrinous networks in severe cases. It therefore becomes hazy, forming a milky "flare" in the beam of the slit-lamp which, as it traverses the anterior chamber, should be invisible (Fig. 106) ; this turbidity interferes with a clear view of the iris and is easily mistaken for haziness of the cornea. In very intense cases polymorphonuclear leucocytes are poured out and sink to the

bottom of the anterior chamber to form a hypopyon. Hyphæma, or blood in the anterior chamber, rarely occurs.

At the same time the nutrition of the corneal endothelium becomes affected so that the cells become sticky and may desquamate in places. There the exudates tend to stick, forming *keratic precipitates* ("keratitis punctata"). These are seldom present in simple iritis, but form an important feature of cyclitis (Fig. 104).

The exudates poured out by the iris and ciliary body also cover the surface of the iris as a thin film and spread into, and sometimes completely over, the pupillary area. When these are profuse the iritis is termed *plastic*. In this manner the pupil may become "blocked," a condition which seriously impairs the sight. Moreover, the exudates tend to stick the iris to the lens capsule so that it becomes fixed. If atropine is instilled at an early stage the iris may be freed and the pupil again becomes dilated and circular. In such cases spots of exudate or pigment derived from the posterior layer of the iris may be left permanently upon the anterior capsule of the lens, forming valuable evidence of previous iritis (Plate VIII, Fig. 1); these are easily distinguished from the congenital spots due to persistence of the pupillary membrane (p. 259). If, however, the adhesions are allowed to become organized they are converted into fibrous bands which atropine is unable to rupture; such firm adhesions of the pupillary margin to the lens capsule are called *posterior synechiæ* (*συνέχειν*, to hold together); they show some predilection for the lower part of the pupil in the early stages, probably owing to gravitation of the plastic exudates. When they are localized and a mydriatic causes the intervening portions of the circle of the pupil to dilate, the pupil assumes a festooned appearance (Plate VIII, Fig. 1). Such an irregular pupil, intensified by the instillation of a mydriatic, is a sign of present or past iritis. Owing to the contraction of organizing exudates upon the iris the pigment epithelium on its posterior surface may be pulled around the pupillary margin so that patches of pigment may be seen on the anterior surface of the iris (*ectropion of the uveal pigment*).

In severe cases of plastic iritis or after recurrent attacks, the whole circle of the pupillary margin may become tied down in this way to the lens capsule. The condition is called *annular* or *ring synechia* or *seclusio pupillæ* (Figs. 185, 186); it is one of great danger to the eye, since, if unrelieved, it inevitably leads to secondary glaucoma. The aqueous, unable to pass forwards into the anterior chamber, collects behind the iris, which becomes bowed forwards like a sail, a condition which is called *iris bombé* (Fig. 185). Regarded from in front, the anterior chamber is seen to be funnel-shaped, deepest in the centre and shallowest at the periphery. The filtration angle is thus obliterated by the apposition of the iris to the cornea at the periphery where eventually adhesions may form (*peripheral anterior*

synechiæ). The circulation of the aqueous is therefore obstructed and the ocular tension rises (p. 286).

When the exudate has been more extensive it may organize across the entire pupillary area which becomes ultimately filled by a film of opaque fibrous tissue ; this condition is called blocked pupil, or *occlusio pupillæ* (Fig. 185). If there has been much cyclitis the posterior chamber also becomes filled with exudates which may organize, tying down the iris to the lens capsule ; this condition of *total posterior synechia* leads to retraction of the peripheral part of the iris, so that the anterior chamber becomes abnormally deep at the periphery, sometimes deeper than in the centre (Fig. 186).

In the worst cases of *plastic iridocyclitis* a cyclitic membrane may form behind the lens where it may be seen with the ophthalmoscope or even by oblique illumination. In young children the condition

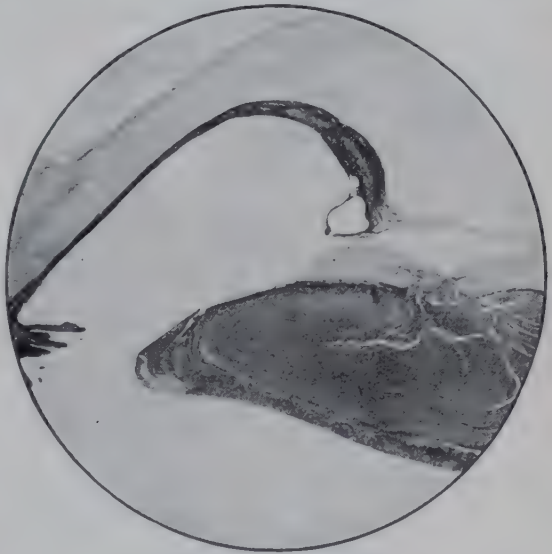


FIG. 185. Extensive anterior peripheral synechia where the iris adheres to the cornea. Posterior synechia with *secusio pupillæ* ; inflammatory pupillary membrane with *occlusio pupillæ*.

forms one type of *pseudo-glioma* (p. 369). In the later stages the degenerative changes in the ciliary body prevent it from fulfilling its functions of supplying the eye with intra-ocular fluid and nutriment. The vitreous suffers first, becoming fluid, and later the lens, which becomes opaque. Finally the eye shrinks (*phthisis bulbi*).

During the course of any type of the disease the *ocular tension* may be affected. Most frequently in the active stages this involves a rise determined, in the first place, by the height of the pressure in the widely dilated capillaries and, in the second, by the difficulty experienced by the sticky albuminous aqueous in escaping through the filtration channels at the angle of the anterior chamber (*hypertensive iridocyclitis*). In the later stages when the pupil has been bound down blocking the flow of aqueous from the posterior to the anterior chamber, a secondary glaucoma may also follow. Finally, if the ciliary region becomes atrophic, interference with the

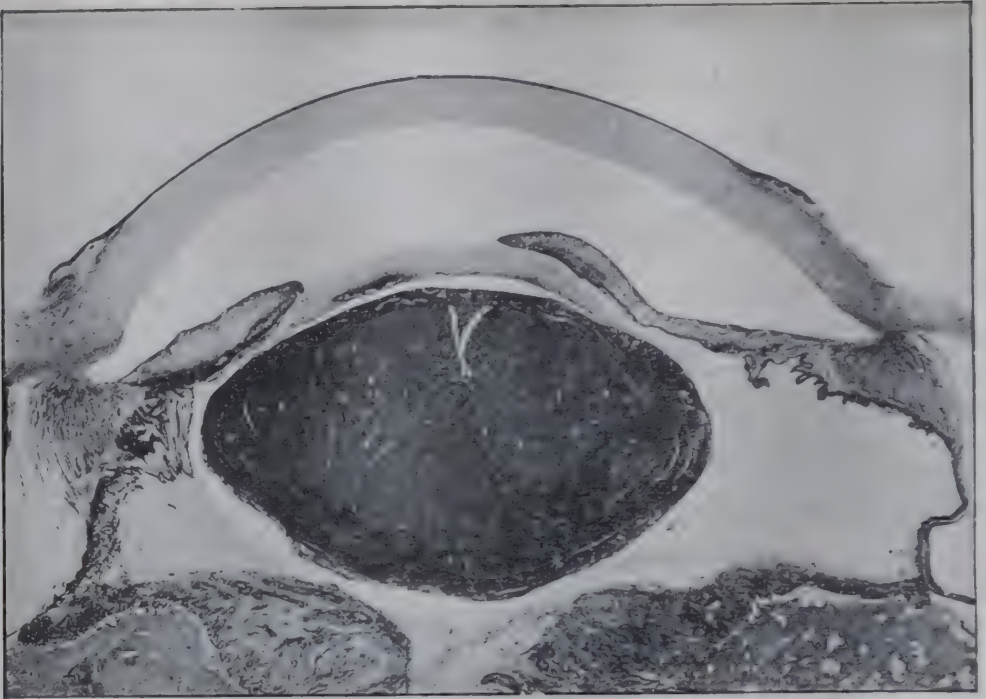


FIG. 186. Total posterior synechia ($\times 7$), from a case of plastic iridocyclitis beginning to cause phthisis bulbi. The iris is completely adherent to the lens capsule, and the periphery is retracted. There is a delicate inflammatory pupillary membrane (occlusio pupillæ). There is also an anterior capsular cataract, due to inflammation ; it contains calcareous deposits, as shown by the patch of dark staining. The ciliary body is degenerated and detached from the sclera at the posterior part. The retina is completely detached and folded behind the lens.

secretion of aqueous may lead to lowering of the ocular tension and the development of a soft eye—an ominous sign.

The Diagnosis of Acute Iridocyclitis. Iritis is most frequently mistaken either for conjunctivitis or acute glaucoma. The error of mistaking iritis for acute glaucoma is the most serious which can be made, particularly because the treatment of the two conditions is diametrically opposed. Dilatation of the pupil with atropine, which is urgently necessary in iritis, is the worst possible treatment of closed-angle glaucoma. At the cost of some repetition, therefore, the distinguishing features will be given here.

(1) *Injection.* Superficial in conjunctivitis, deep ciliary in iritis and glaucoma.

(2) *Secretion.* Muco-purulent in conjunctivitis ; watery in iritis and glaucoma.

(3) *Pupil.* Normal in conjunctivitis ; small and irregular in iritis ; large and oval in glaucoma.

(4) *Media.* Clear in conjunctivitis ; sometimes opacities in the pupil in iritis ; corneal oedema in glaucoma.

(5) *Tension.* Normal in conjunctivitis ; usually normal in iritis ; raised in glaucoma.

(6) *Pain.* Mild discomfort in conjunctivitis ; moderate in eye and first division of trigeminal in iritis ; severe pain in eye and the entire trigeminal area in glaucoma.

(7) *Tenderness.* Absent in conjunctivitis ; marked in iritis and glaucoma.

(8) *Vision.* Good in conjunctivitis ; fair in iritis ; poor in glaucoma.

(9) *Onset.* Gradual in conjunctivitis ; usually gradual in iritis ; sudden in glaucoma.

(10) *Systemic complications.* Absent in conjunctivitis ; little in iritis ; prostration and sometimes vomiting in glaucoma.

Cyclitis has already been referred to incidentally. In the severe *plastic* form the exudates from the ciliary body pass into the anterior chamber directly from that part which forms a boundary of the chamber (Fig. 8), and indirectly by passing forwards through the pupil. The deposition of keratic precipitates on the back of the cornea is a prominent feature, while clouds of dust-like opacities appear in the vitreous. When the exudates organize they not only cause a total posterior synechia, but also surround the lens and extend throughout the vitreous. Behind the lens they form a transverse membrane or *cyclitic membrane*. Strands of fibrous tissue are formed in the vitreous which become anchored to the retina in various places, and by their subsequent contraction may lead to detachment of this tissue. The exudates which organize upon the surface of the ciliary body cause the destruction of the ciliary processes, which results in diminishing or abolishing the secretion of aqueous. Hence the intra-ocular tension becomes lowered (*hypotony*) and the eye may even become shrunken and quadrilateral

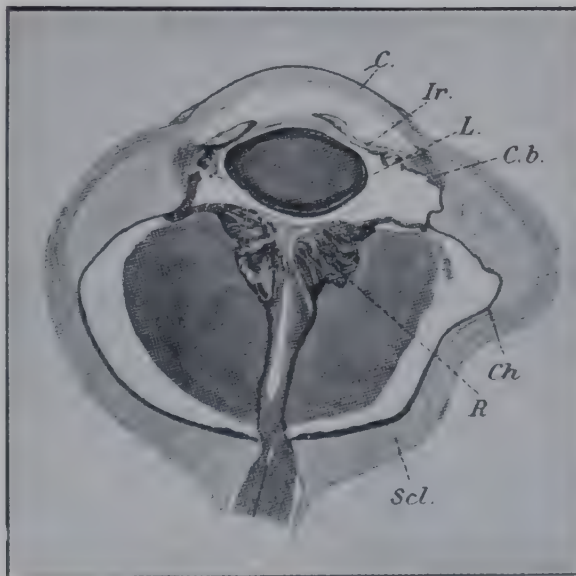


FIG. 187. Phthisis bulbi, due to iridocyclitis ($\times 3$).
C., cornea ; *Ir.*, iris ; *L.*, lens ; *C.b.*, ciliary body ;
Ch., choroid ; *R.*, retina, detached and folded up
 behind lens, separated from choroid by albuminous
 coagulum ; *Scl.*, sclera.

in shape owing to pressure by the rectus muscles—*phthisis bulbi* (Fig. 187) ; thereafter degenerative changes supervene.

Chronic iridocyclitis (*Simple cyclitis*) deserves special mention because of its insidiousness and the difficulty in its diagnosis. It is an extremely chronic disease characterized by diminution of vision with few physical signs. In severe cases there is some ciliary congestion, tenderness on pressure over the ciliary region, a deep anterior chamber, keratic precipitates on the back of the cornea and dust-like opacities in the vitreous. The keratic precipitates indeed, may be the only obvious evidence of the disease, sometimes scattered over a triangular area of the lower part of the cornea, an arrangement due to convection currents in the aqueous and gravitation of the particles towards the bottom of the anterior chamber (Plate VIII, Fig. 2), but more commonly scattered irregularly over the lower part of the cornea as a few isolated spots. They require great care in examination for their discovery (p. 102) and their importance cannot be over-estimated. The smaller spots sometimes coalesce forming small plaques which gradually become translucent ("mutton-fat k.p.").

The vitreous opacities are of the same nature ; they are mainly wandering leucocytes but many are coagulated fibrin and particles of albuminous exudate. Their mobility in the vitreous shows that this gel has become fluid.

In the most insidious cases the symptoms and physical signs are minimal. Considerable diminution of vision without obvious cause should always excite apprehension, and the cornea should be most carefully explored with the slit-lamp. A few spots of "k.p." are decisive proof of cyclitis and may be the sole physical sign. A change in the colour of the iris due to atrophy is an important sign since it may at once attract attention, especially if the normal eye has a brown iris ; it indicates, however, a late stage of the disease.

The disease is generally very chronic and liable to exacerbations with the gradual and insidious formation of posterior synechiæ. Vision is diminished during the more acute phases and recovers considerably in the intervals, but each recurrent attack leaves a more permanent defect. The eye may finally become soft and tender and enter into the condition of *phthisis bulbi*, but this occurs only after many years.

An important and not uncommon complication is a rise of tension in the course of the disease to constitute the clinical syndrome of *hypertensive iridocyclitic crises* (of Posner and Schlossman). In this condition the eye may appear normal but periodically acute or subacute attacks of raised tension occur associated with the presence of an aqueous flare and keratic precipitates, often so few and unobtrusive as only to be seen by examination with the slit-lamp and often of relatively short duration. These attacks, accompanied by the diminution of vision and the appearance of halos around lights

PLATE VIII

INFLAMMATIONS OF THE UVEAL TRACT

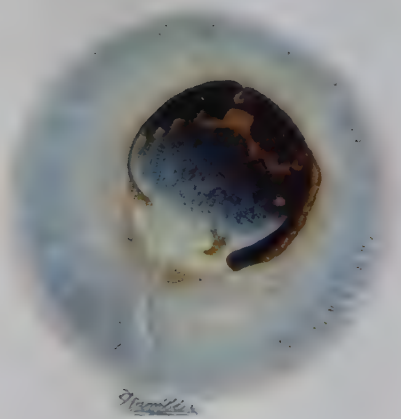


FIG. 1. Plastic iritis.



FIG. 2. Chronic cyclitis.



FIG. 3. Panophthalmitis.



FIG. 4. Pseudo-glioma.



FIG. 5. Phthisis bulbi.

[To face p. 234.]

PLATE IX
CHOROIDITIS



FIG. 1. Miliary tuberculosis (acute stage).

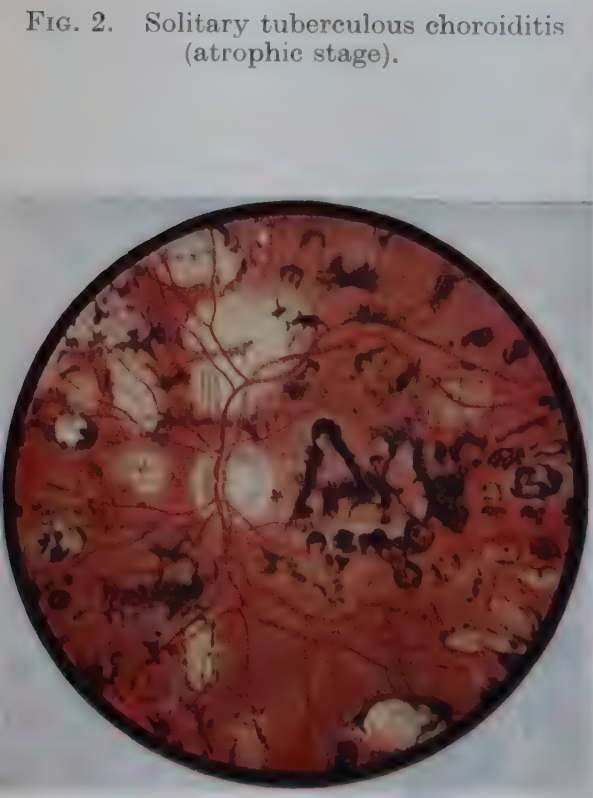


FIG. 2. Solitary tuberculous choroiditis (atrophic stage).



FIG. 3. Disseminated syphilitic choroiditis (atrophic stage).

with headaches, resemble so closely attacks of closed-angle glaucoma that a mistake in diagnosis is often made. The diagnosis may depend solely on the detection of one or two keratic precipitates, but its establishment is of the utmost importance since in this condition treatment by atropine with topical corticosteroids rapidly resolves the attack, a method of therapy which, of course, would be disastrous in a case of closed-angle glaucoma. These recurrent attacks although controlled by atropine, tend to recur until the cause of the inflammation is eliminated.

Chronic posterior cyclitis, affecting essentially the pars plana of the ciliary body and the periphery of the choroid, occurs particularly in children and young adults. The onset is insidious without dramatic evidences of inflammation and the first symptom is a deterioration of vision due to opacities in the anterior vitreous. The inflammation is non-specific, the cause is usually unknown, and after a long course the results are frequently serious. Treatment must be on general lines.

Varieties of Anterior Uveitis

While the general description of the course of acute and chronic non-purulent anterior uveitis applies to the majority of cases the ætiology of which is undetermined, a large number of different types are met with clinically. In the most general sense these may be divided into two main varieties—those due to direct organismal

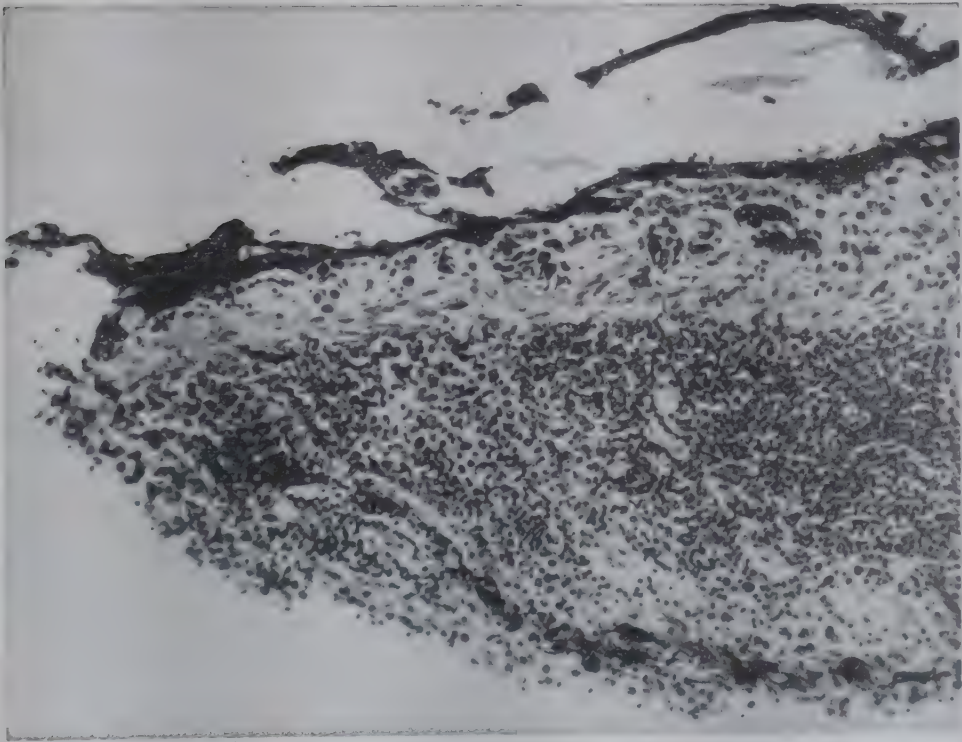


FIG. 188. Granulomatous iridocyclitis (in brucellosis). A nodule of lymphoid cells near the sphincter of the iris (A. C. Woods).

infection and those which are an expression of an allergic reaction. The clinical pictures presented by infections with specific organisms will be discussed at a later stage.

Infective (granulomatous) iridocyclitis is due essentially to the invasion of the eye by non-purulent organisms. In the absence of allergic sensitivity of the tissues, the inflammation tends to be insidious in onset and chronic in course with a minimum of inflammatory reaction. Such chronic inflammations due to direct

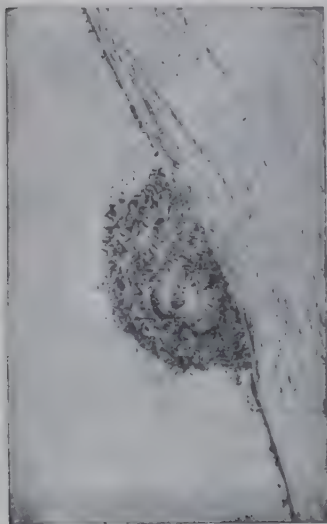


FIG. 189. Corneal precipitate in tuberculous cyclitis.

organismal infection are typically characterized by dense nodular infiltrations of the tissues rather than by diffuse exudative phenomena (Fig. 188). In the absence of immunity these lesions show a slow and gradual extension; if immunity is present they tend to become circumscribed and relapse if the resistance falls. If hypersensitivity develops a further organismal invasion leads to necrosis and caseation. Clinically such an inflammation is characterized by the formation of dense synechiæ and large greasy "mutton-fat" keratic precipitates composed of endothelial cells and lymphocytes (Fig. 189). The clinical picture varies considerably with the type of organism concerned; the more important manifestations will be considered presently.

The *allergic (exudative) type of reaction*, on the other hand, tends to be of acute onset and short duration, diffuse in extension and without focal lesions in the iris. There is a considerable flare in the anterior chamber and the keratic precipitates, if present, are few and composed of lymphoid cells.

The course of the disease varies. Milder cases take three or more weeks before the inflammation subsides; improvement is shown by good dilatation of the pupil with atropine, diminution of injection and pain. In the chronic cases the ciliary body is always seriously involved and the inflammatory signs are less. Complete resolution may occur in mild cases treated early and suitably, particularly if early dilatation of the pupil has forestalled the development of posterior synechiæ. In all cases, however, a characteristic feature is the tendency to relapse. Each fresh attack runs a similar course, although usually less severe, often leaving further traces and increased impairment of vision. Repeated attacks of iritis lead to atrophy of the iris, which assumes a dirty grey colour like felt or blotting paper. Red streaks often mark the site of permanently dilated vessels, usually of new formation and therefore not necessarily radial in direction. The pupillary margin is thin and frayed; the reactions are diminished.

The General Course of (Non-purulent) Posterior Uveitis

Inflammations of the choroid conform in their general characteristics with those affecting the anterior part of the uveal tract ; they may either appear in the form of isolated foci of inflammation or they may be diffuse ; in the latter case the anterior uvea is always involved. It is to be remembered that the outer layers of the retina are dependent for their nutrition upon the choroid so that an inflammation of the latter always involves the former secondarily. Primary affections of the retina may occur without involvement of the choroid : primary affections of the choroid invariably involve the retina in greater or less degree.

Non-suppurative Choroiditis may occur in two forms. As with iridocyclitis, a *granulomatous* form is associated with direct organismal infection, the essential feature of which is the occurrence of localized accumulations of chronic inflammatory cells (lymphocytes, plasma cells, etc.). This form of choroiditis will be discussed at a later stage.

Exudative choroiditis, on the other hand, is a non-specific plastic inflammatory response characterized initially by more acute cellular infiltration (leucocytes) and much exudation, the ætiology of which is exactly comparable with the similar type of iridocyclitis.

The Course of Exudative Choroiditis. A recent focus is seen ophthalmoscopically as a yellowish area ; when near a retinal vessel it lies at a level deeper than the vessel. It is due to infiltration of the choroid, the exudates hiding the choroidal vessels which cause the normal red reflex. In the early stages the membrane of Bruch is intact ; in these circumstances only fluid can pass through it, but this suffices to make the overlying retina cloudy and grey so that the edges are hazy and ill-defined. The exudates not only pass into, but also through the retina, so that punctate or diffuse opacities are seen in the vitreous. In the later stages the membrane of Bruch may be destroyed, although like all elastic membranes it offers considerable resistance ; when this has occurred leucocytes are enabled to pass through into the retina and vitreous (Fig. 190). A marked vitreous haze usually indicates that the ciliary body is also involved ; while the presence of keratic precipitates on the back of the cornea and inconspicuous posterior synechiæ proves that in many cases of apparently localized choroiditis the whole uveal tract is implicated (general uveitis).

Owing to the fibroblastic activity of the choroidal stroma the exudates become organized, so that fibrous tissue is formed which destroys the normal structures and fuses the choroid and retina firmly together. The colour of the spots therefore gradually changes to white, partly due to the fibrous tissue deposited, partly to

thinning and atrophy whereby the white reflex from the sclera is permitted to shine through (Plate IX, Fig. 3).

The pigment of the retinal pigmentary epithelium is extremely resistant, even although the cells which contain it are destroyed. It tends to become heaped up into masses, partly intra-, partly extra-cellular : moreover, the pigment cells are stimulated to proliferate. Isolated masses of black pigment are thus formed in the white areas, especially at the edges, so that in the atrophic stage the white areas are surrounded by a black zone of pigment (Plate IX, Fig. 3). The process has then reached its natural

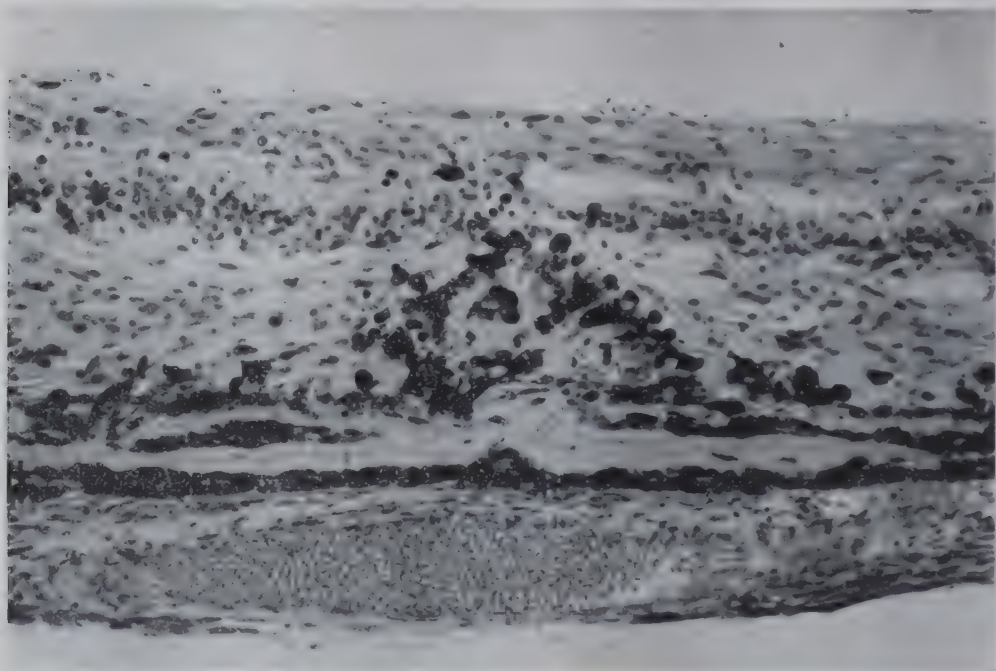


FIG. 190. Chorioretinitis. A patch of chorioretinitis showing scarring of the retina and choroid with fusion between the two tissues and proliferation of the pigmentary epithelium (Ashton).

termination, and these sharply defined areas remain permanently unaltered.

The symptoms in the early stages are principally the defects of vision due to the retinal lesions and to cloudiness of the vitreous ; they are thus marked when the lesion is in the central area and usually escape observation when in the periphery. The inflamed area is slightly raised, so that the contour of the retina is altered. This causes distortion of the images, giving rise to a similar appearance of distortion of the objects seen—*metamorphopsia* : thus straight lines appear to be wavy. Frequently objects appear smaller than they are—*micropsia* ; sometimes larger—*macropsia* : these effects are due to separation or crowding together respectively of the rods and cones. Subjective flashes of light due to retinal

irritability (*photopsiæ*) are seen. These subjective symptoms are often accompanied by the perception of a black spot in front of the eye, corresponding to the lesion—a *positive scotoma*.

In the later stages the affected areas are incapable of giving rise to visual impulses so that *negative scotomata* exist in the field of vision, wherein there is a hiatus in the field of vision of the same nature as the normal blind spot. Their importance depends upon their situation. Peripheral scotomata may pass unnoticed, a central scotoma destroys direct vision; in the latter case peripheral



FIG. 191. Recurrent patches of choroiditis showing a juxtapapillary lesion above and to the left, an old lesion to the right of the disc and two large and a minute patch towards the periphery.

vision still permits the patient to get about, but all fine work is impossible.

The disease is chronic, organization of the exudates taking several weeks. The occurrence of fresh spots may extend the acute stage over a period of months, and the ultimate defects are permanent. The condition is often bilateral (Fig. 191).

Non-suppurative choroiditis is usually classified according to the number and location of the areas involved.

Disseminated Choroiditis. In this type, small areas of inflammation are scattered over the greater part of the fundus behind the equator (Plate IX, Fig. 3). In the milder cases only

a few spots are formed and the exudates in the vitreous become absorbed. In the more severe the spots are very numerous, fresh foci arising and passing through the same stages, until finally the whole fundus may be covered with atrophic areas passing through the stages of evolution just described. The vitreous opacities increase, and finally the nutrition of the lens suffers and a complicated cataract results. Owing to the transience of the acute stage the atrophic stage naturally comes much more frequently under observation. Such a condition may be due to syphilis, sometimes acquired, sometimes congenital, and hence may be associated with interstitial keratitis; other cases are tuberculous but in many the cause is obscure. The changes produced by myopia cause similar signs and symptoms but they are degenerative in nature.

Anterior choroiditis is often syphilitic, and manifests itself in the same form as disseminated choroiditis, but is confined to the peripheral parts of the fundus. On this account it is frequently symptomless and is discovered only on routine examination. It should be clearly distinguished from the similar changes found in high myopia and in old people as a senile degeneration.

Central choroiditis occurs in disseminated choroiditis, and occasionally alone.

Juxtapapillary choroiditis (Jensen) occurs in young persons as an exudation close to the disc, usually oval in shape and about the same size as the disc (Fig. 191). The exudates may cover the retinal vessels, and there are vitreous opacities and sometimes "k.p." There is generally a sector-shaped defect in the field of vision. The cause is usually obscure. The inflammation slowly subsides, leaving a patch of atrophy, but recurrence may take place.

Diffuse choroiditis is characterized in the early acute stage by one or more plaques of yellowish white or grey areas, shading off at the edges into normal fundus. The patches spread and coalesce so that the greater part of the fundus may be finally involved but islands of normal fundus usually survive. The exudates organize, leaving white areas in which the larger choroidal vessels persist as a characteristic band-like network. The retinal pigment becomes heaped into black, irregular spots, over which the retinal vessels course little changed in appearance. Fresh exudates occur simultaneously with the organization of older ones, and sometimes the edge of a patch appears to creep over the fundus like the advance of a myxomycetes. Some of these cases are syphilitic, some tuberculous; in others the cause cannot be traced, but probably some are due to metastatic bacterial invasion.

The General Treatment of Uveitis

In *anterior uveitis*, dilatation of the pupil with atropine, hot applications, and the control of the acute phases of the inflammation, if necessary, with steroids, are the essentials of local treatment.

Atropine acts in three ways : (1) by keeping the iris and ciliary body at rest ; (2) by diminishing hyperæmia ; (3) preventing the formation of posterior synechiæ and breaking down any already formed. It may be used in the form of drops or ointment (1 per cent.) which should be pushed in the early stages, better by frequency of application rather than by increased strength ; every four hours is usually sufficient. When the pupil is well dilated, twice a day suffices. A more powerful initial effect may be obtained by placing a crystal of the alkaloid in the lower fornix. If atropine irritation ensues one or other of the substitutes for this drug (p. 40) may be used. A very powerful mydriatic effect is also obtained by the subconjunctival injection of 0.3 ml. of mydracaine (p. 563), a mixture of atropine, procaine, and adrenaline.

Hot applications are extremely soothing to the patient by diminishing the pain, and are of therapeutic service in increasing the circulation. Hot fomentations and bathings may be used, but dry heat or medical diathermy is often very effective (p. 564).

Corticosteroids (p. 145) administered as drops, ointment, or—more effectively—as subconjunctival injections are of great value in controlling the inflammation in the acute phases in many but by no means all cases. Occasionally the results are dramatic and the eye becomes white with great rapidity. It must be remembered, however, that it merely blankets the inflammatory reaction without affecting the causal factors or curing the disease, so that, unless the cause be removed, relapses are certain to occur on the cessation of treatment. While not therefore in any sense curative, they relieve symptoms and avoid the complications of severe intra-ocular inflammation. In chronic cases they are virtually valueless.

Aspirin is very useful in relieving pain, but if it is intense morphia preparations may be indicated. In the convalescent stage dark glasses are ordered—for both eyes, on account of the consensual reaction to light. Atropine, or its equivalent, should be continued for at least ten days to a fortnight after the eye appears to be quiet, otherwise a relapse is likely to occur.

The treatment of *choroiditis* is unsatisfactory since gross damage is usually done to the retina before the condition can be controlled. The essentials of local treatment are rest in bed, the administration of atropine and heat, either dry by diathermy or an electric pad, or by hot compresses (p. 570). The local use of the sulphonamides or antibiotics and of corticosteroids is usually disappointing ; but sometimes the systemic administration of this hormone or ACTH cuts short an attack and hastens healing (Chapter 14).

Systemic treatment of both anterior and posterior uveitis is directed towards maintaining the general health ; rest is necessary, the bowels should be kept freely open and an attempt should be made to attack the cause of the condition.

In the nondescript forms of uveitis a search should be made for

any obvious septic focus in the body and the result anticipated, particularly if a streptococcal origin is suspected, by a course of one of the sulphonamides or antibiotic drugs (Chapter 14). If the response is not good, or where no satisfactory cause can be found, salicylic preparations may be beneficial in acute cases. In the present unsatisfactory state of our knowledge of the ætiology of most of these cases, obvious septic foci may be radically treated when possible, but always with care lest more harm be done than good; the results, however, are rarely dramatic. In some chronic cases which show little response, treatment by protein shock may produce a good effect (p. 147). Specific measures for the treatment of individual infections will be discussed at a later stage.

Treatment of Sequelæ and Complications

Secondary glaucoma forms one of the most serious complications of iridocyclitis. If this condition of *hypertensive iridocyclitis* develops in the early stages of the disease before the formation of anatomical hindrances to the circulation of the aqueous humour (posterior or peripheral synechiæ), the most effective treatment is to intensify atropinization in order to allay the inflammatory congestion. The use of eserine merely accentuates the irritative state of the circulation. Corticosteroids administered topically and acetazolamide given systemically are frequently very useful in such cases.

If this does not relieve the condition a paracentesis may be performed (p. 413), but only if these hypotensive measures are unsuccessful. The immediate result is often satisfactory since the eye is flushed with plasmoid aqueous containing antibodies, but the paracentesis may require to be re-opened while all the time full atropinization is maintained. Repeated paracenteses, however, should only be done with reservations lest anterior peripheral synechiæ develop; if a good result is not soon obtained an iridectomy may be indicated. It may be necessary to perform a permanent drainage operation but this should only be done as a last resort since the results are often disappointing.

Annular synechiæ demand an iridectomy in all cases in order to restore communication between the posterior and anterior chambers, and thus avoid the supervention of secondary glaucoma. In some cases it is necessary or advisable to be content with making a puncture or transfixion of the iris by a broad needle; at other times an iridectomy is necessary (p. 415). No operative procedure of this kind must be undertaken during an acute attack of iritis if it can be avoided, since the traumatic iritis set up will frustrate the object of the operation by filling the opening with exudates. It is best, if possible, to forestall a ring synechia by performing the iridectomy before the adhesion extends round the whole circle. This can often be done because a ring synechia is frequently the result of recurrent attacks and can therefore be performed during a quiescent interval.

It is often difficult to get a good gap owing to the atrophy and friability of the iris and the firmness of the adhesions ; hæmorrhage is common. Iridectomy sometimes has a beneficial effect on recurrent iritis, but should not be done without special indications until all other non-operative measures have failed. The presence of "k.p." should generally be regarded as a contra-indication to surgery in the absence of dangerously high intra-ocular pressure.

Total posterior synechia can seldom be operated upon with success. Iridectomy is seldom possible, and the only procedure which can be adopted is extraction of the lens by a specially devised and somewhat hazardous operation wherein, after retroflexing the cornea with a suture to expose freely the operative field, the synechiæ are ruptured as far as possible with an iris repositor, a sufficiency of iris and organized scar tissue is cut away and the lens then extracted.

If, finally, the globe is blind and shrunken and particularly if it is painful and a continual source of annoyance to the patient, it should be excised.

Purulent Uveitis and Panophthalmitis

Purulent uveitis is generally caused by infected wounds, whether accidental or the result of operations or ulcers. In the case of ulcers and when the penetrating wound is corneal, the inflammation may remain as an anterior uveitis if the infection is not virulent or if it is controlled by treatment ; but the usual tendency is for the whole eye to be involved in a *panophthalmitis* (Plate VIII, Fig. 3). In deeper injuries the vitreous is usually first affected ; organisms grow readily in it as in a culture medium, and purulent cyclitis, retinitis and choroiditis develop (Fig. 192). The pneumococcus is responsible for many cases, so also are the staphylococcus, the streptococcus, *E. coli*, *Ps. pyocyanea*, *B. cereus (subtilis)*, and *Cl. welchii* (gas gangrene).

The endogenous form is metastatic in origin and may arise from an infective embolus of a retinal artery or of a choroidal vessel ; whatever the origin, an intense uveitis occurs involving all the tissues of the eye, which progresses in the same manner as an exogenous panophthalmitis. Such cases are now seldom seen, but occur in the course of infectious diseases, especially pneumonia, influenza, measles, scarlet fever, meningitis or furunculosis. In this group the condition may be bilateral.

In both forms there is rise of temperature, headache, and sometimes vomiting. In the exogenous forms the edges of the wound become yellow and necrotic, a hypopyon appears, there is great chemosis with intense ciliary and conjunctival congestion, and the lids are swollen and red. There is severe pain in the eye, due at first to iritis, later to increased tension. The vitreous becomes purulent, as shown by a yellow reflex with oblique illumination. The anterior chamber soon becomes full of pus, and the cornea cloudy and yellow ;

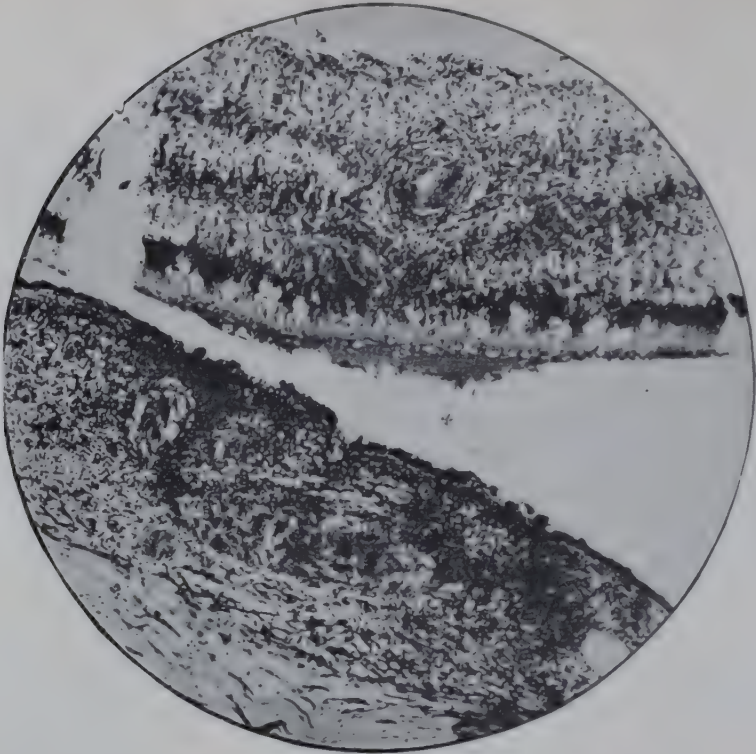


FIG. 192. Section of the retina (above) and choroid in panophthalmitis following a perforating wound ($\times 60$).

ring infiltration may occur (p. 199). There may be proptosis and limitation of movement of the globe due to extension of the inflammation to Tenon's capsule.

In metastatic cases, ophthalmoscopic examination shows that the media are hazy so that the yellow oedematous retina is only dimly seen (Fig. 193) ; there is a yellow reflex, the formation of a hypopyon and rapid failure of vision.

In the less severe cases or when the condition is controlled at an early stage by treatment, the inflammation may resolve. There may be some development of cyclitic fibrous tissue in the vitreous and some scarring of the retina in which atrophic areas accompanied by pigmentary disturbances persist. In some cases the optic disc has been the chief local focus and a clinical picture resembling intense papillitis is seen with much exudation extending into the neighbouring retina.

In more severe cases the inflammation gives rise to the widespread formation of cyclitic membranes, destruction of the ciliary processes and a fall in ocular tension resulting in shrinkage of the globe. In the most severe cases and when the infection is allowed to take its course, the pus bursts through the walls of the globe, usually just behind the limbus ; thereupon the pain subsides and after prolonged suppuration the eyeball shrinks (Plate VIII, Fig. 5). The prognosis

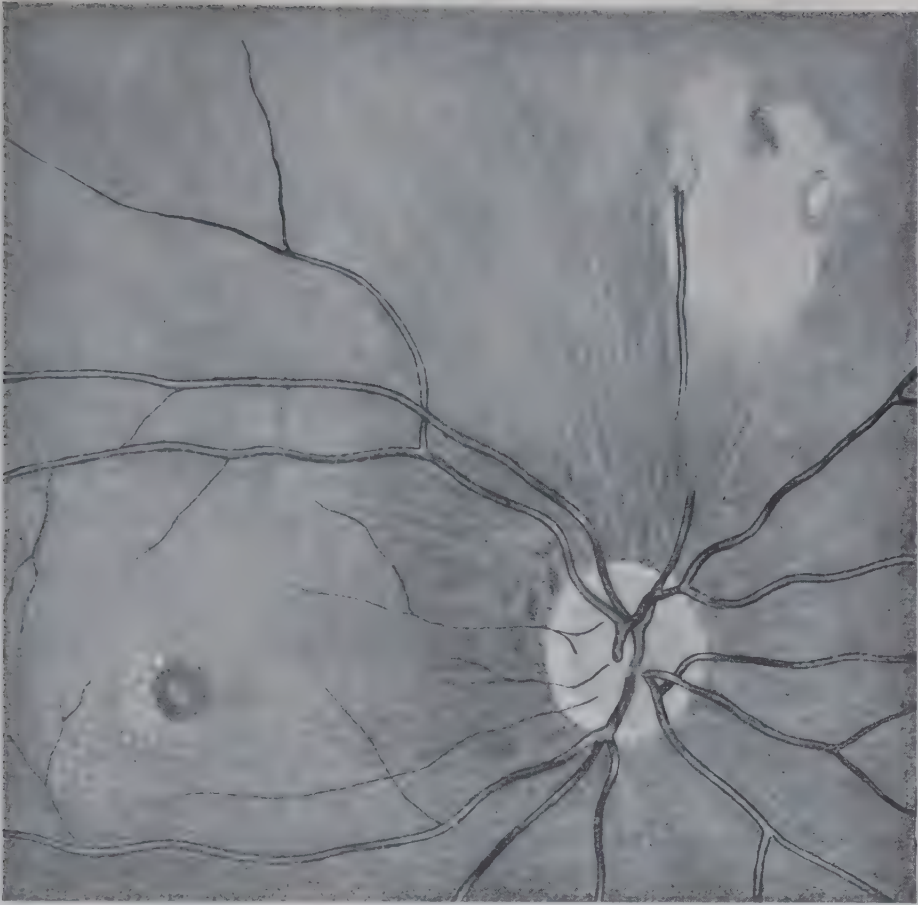


FIG. 193. Metastatic choroiditis.

is bad, for vision is almost invariably lost. The condition is not likely to set up sympathetic ophthalmitis.

A subacute form occurring in children takes a plastic course characterized by the exuberant development of fibrous tissue of cyclitic origin. This gives rise to one form of so-called "*pseudo-glioma*" (Plate VIII, Fig. 4). It is often first noticed by the mother as a whitish reflex in the pupil (amaurotic cat's eye). Generally there is no knowledge of inflammation having occurred in the eye, but a history may be obtained of fits, aural disease, an acute specific fever, basal meningitis or some other serious illness. Several cases have been proved by pathological examination to have been due to the meningococcus. There are usually signs of past iritis or iridocyclitis. The diagnosis from retinoblastoma is of great importance (p. 369).

Treatment. In the early stages after wounds whether operative or accidental, an attempt must be made to stay the process. Treatment by antibiotics, both local and systemic, should be used to its fullest extent at the earliest possible moment, the choice of drug depending on the organism involved (Chapter 14). Until it is identified—or in many cases wherein identification is impossible—penicillin or soframycin may be injected subconjunctivally every three or four hours; in desperate cases an intra-ocular injection

of a solution of the pure salt of penicillin may be made into the anterior chamber or even the vitreous. Sulphonamides or an anti-biotic which enters the eye, such as chloramphenicol, should be given systemically in full doses. Local treatment by steroid preparations (Chapter 14) should also be intensively used. Hot bathings and medical diathermy are applied, and atropine instilled. In many cases the eye may be saved by these measures if applied early and intensively ; but too often vision is lost.

As soon as it is evident that the eye cannot be saved it should be *excised* or, if there is any danger of the escape of pus during the operation, *eviscerated* (*q.v.*). In most cases a very satisfactory operation which allows more rapid healing than an evisceration and at the same time prevents a spread of the infection up the optic nerve sheath which might give rise to a meningitis, is a *frill excision* (*q.v.*) whereby a collar of sclera is left around the optic nerve.

Specific Types of Uveitis

The more important types of uveitis associated with specific infections will now be discussed.

Syphilis may attack any part of the uveal tract and the clinical manifestations of the infection are protean.

Syphilitic iritis manifests itself in two forms. A *simple plastic iritis* occurs typically in the secondary stage of the disease, soon after the skin eruptions, usually within the first year after infection, but not before the third month. There is usually nothing characteristic about this form of iritis ; it lasts from two to eight weeks and does not usually recur. In the absence of early anti-syphilitic treatment it is seen in at least 3 to 4 per cent. of syphilitics, usually in males, and is generally unilateral, but the second eye may become affected later. The Wassermann reaction is of great value in settling the diagnosis ; the *Treponema pallidum* has been found in the aqueous.

A plastic iritis also occurs in congenital syphilis, usually as an accompaniment of interstitial keratitis (*q.v.*). It also occurs in very young babies with congenital syphilis without any corneal complication, but usually with large nodules or gummata on the iris. The average age of onset is five to six months but it is sometimes seen so soon after birth as to suggest an intra-uterine inflammation. It is commoner in females than in males, and is unilateral in about half the cases.

A plastic iritis may occur late in the disease ; it is usually mild but recurrent and may thus be serious owing to cumulative damage.

Finally, an acute plastic iritis may occur as a *Herxheimer reaction* twenty-four to forty-eight hours after the first therapeutic dose of arsenic or penicillin, due probably to the flooding of the system with treponemal toxins.

Gummatous iritis occurs late in the secondary or rarely during the tertiary stage. These cases are characterized by the formation of yellowish-red, heavily vascularized nodules near the pupillary and ciliary borders of the iris, but not in the intermediate region ; they are usually multiple and vary in size from that of a pin's head upwards and are generally associated with much exudation and broad synechiæ. The nodules are liable to be mistaken for tubercles (*q.v.*) or a neoplasm (*q.v.*). A gumma may rarely extend to the corneo-sclera at the angle of the anterior chamber and lead to perforation of the globe. The sites of previous gummatous deposits in the iris are marked by depigmentation of the stroma so that whitish spots appear in the ciliary region of the iris or a circle of atrophic patches near the pupillary border. These appearances are strong evidence of syphilis.

A *gumma of the ciliary body* is rare. It causes an intensely acute plastic iridocyclitis, much exudation into the anterior chamber, often deep infiltration of the cornea and usually great pain. It is only to be diagnosed clinically with certainty when the inflammation extends into the sclera (p. 223). If it does not respond to active anti-syphilitic treatment, the eye eventually becomes phthisical.

Vision is usually impaired permanently in syphilitic iritis, and the prognosis is worse in the gummatous type. Specific iritis is a sign of severe syphilis ; Trousseau found that thirty-four out of forty patients ultimately developed grave sequelæ such as tabes and general paralysis.

Syphilitic choroiditis may occur as disseminated choroiditis, peripheral choroiditis or as a diffuse lesion (Plate IX, Fig. 3).

In addition to the usual local measures the patient is treated by systemic penicillin or another suitable drug ; it may be supplemented by iodides.

Gonorrhœa usually limits itself to the anterior uveal tract.

Gonorrhœal iritis is common. A very characteristic form may occur during the acute attack of the disease when a hyphæma may appear or the anterior chamber contain exudates which have a peculiar gelatinous appearance and a greenish-grey colour which is characteristic. There is little doubt that it is a metastatic infection. Extension to the ciliary body may be indicated by fine vitreous opacities, but involvement of the choroid is rare. The patients are almost always men, and as a rule both eyes are affected, although not at the same time. A less characteristic plastic form of iritis occurs especially in those cases which have gonorrhœal "rheumatism" and seldom supervenes until after an attack of arthritis, usually in the knees. The iritis tends to recur persistently, sometimes associated with arthritis, and often many years after the first attack so that extensive synechiæ usually result.

Gonorrhœal choroiditis is rare and non-specific in appearance.

Treatment. An intensive course of sulphonamides in full doses

should be started at once and may well be supplemented by the local administration of penicillin or other antibiotic (Chapter 14). In acute cases subconjunctival injections of penicillin have a dramatic effect. In chronic or recurrent cases a gonococcal vaccine and treatment of the prostate sometimes produce good results.

Tuberculosis. Tuberculosis may affect any part of the uveal tract.

Tuberculous iritis of the metastatic granulomatous type occurs in a miliary and a conglomerate or solitary form. In the *miliary type* there is usually a yellowish-white nodule surrounded by numerous smaller satellites, usually, as in gummatous iritis, near the pupillary or ciliary margin. In the earliest stages the nodules are minute, greyish and translucent, and the presence of "k.p." on the back of the cornea indicates involvement of the ciliary body. Hyphæma is not uncommon; and pseudo-hypopyon, composed of caseating tuberculous material, may occur.

In *conglomerate tubercle* there is a yellowish-white tumour, although smaller satellites may be present; the nodules contain giant-cell systems. There is usually less general iritis than in the miliary form, but there is always some. The condition may be mistaken for gummatous iritis or a neoplasm. The absence of specific history, a negative Wassermann reaction, the failure of anti-syphilitic treatment, and the age of the patients—children or young adults—are features distinguishing it from gummatous iritis. The presence of satellites, the usual but not invariable absence of visible vessels upon the surface of the nodules, the age of the patient, and the presence of iritis distinguish it from a malignant melanoma.

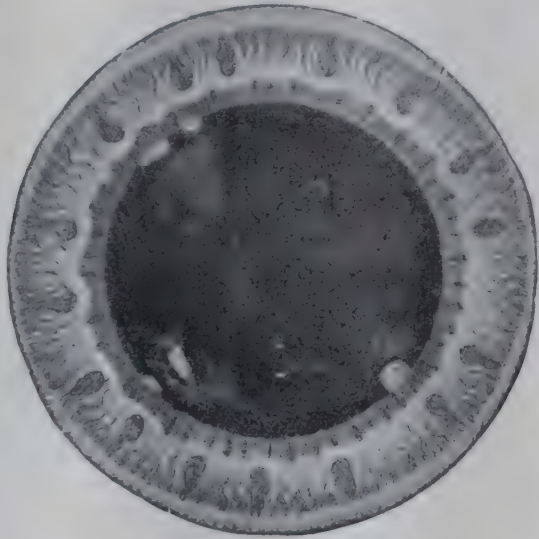
In conglomerate tubercle of the iris the corneo-sclera at the angle of the anterior chamber almost invariably becomes ultimately eroded so that the tuberculous mass grows rapidly through the perforation, and a large portion of the iris may become prolapsed. Such an eye is inevitably lost.

An *exudative type of iritis*, as we have seen, is often considered tuberculous in origin, probably allergic in nature. Clinically there is little to distinguish it from other types of the disease. It is usually of a recurrent or very chronic nature with large "mutton-fat" keratic precipitates (Fig. 189). Occasionally minute, translucent nodules appear on the surface of the iris, especially at the pupillary border (*Koeppé's nodules*) (Fig. 194). These are considered of some diagnostic significance and should be carefully looked for.

Tuberculous choroiditis occurs in acute or miliary and chronic forms. Miliary tubercles are found in acute miliary tuberculosis, and especially in tuberculous meningitis, usually as a late event, but they are occasionally seen before there is any evidence of meningitis or generalized tuberculosis. They used to be common in the late stages of tuberculous meningitis in children. Ophthalmoscopically they appear as round, pale yellow spots, usually near the

disc, although any part of the choroid may be attacked (Plate IX, Fig. 1). Generally only three or four spots are seen, but as many as sixty or seventy have been found. They afford most important diagnostic evidence of tuberculosis in cases of meningitis and

FIG. 194. Tuberculous nodules on the pupillary margin and the anterior lens capsule in tuberculous cyclitis (v. Szily).



obscure general disease. Microscopically they consist of typical giant-cell systems, containing a variable number of tubercle bacilli (Fig. 195). Until the introduction of chemotherapy in this disease, miliary tuberculosis of the choroid was usually a prelude to death, whereas now recovery is common.



FIG. 195. Section of miliary tuberculosis of the choroid ($\times 60$), from a child with tuberculous disease of the hip who died of meningitis. Note the giant cells and the small round cells at the periphery.

Chronic tuberculosis may occur as a diffuse or disseminated inflammation affecting large areas or the whole choroid, and characterized by the extensive development of granulation tissue ; or, more rarely, as a solitary or conglomerate mass simulating a neoplasm, but usually showing definite signs of inflammation such as œdema of the retina or vitreous opacities. The primary systemic focus is rarely, if ever, in the acute stage. Ophthalmoscopically the diffuse form shows areas with hazy edges raised somewhat above the surrounding fundus and covered by œdematous retina. There are usually vitreous opacities. The mass, consisting of granulation tissue containing giant cells, spreads until it involves the retina and may finally fill the posterior part of the globe. Similar ophthalmoscopic appearances are met with resulting from metastatic choroiditis (*q.v.*) and the exudative retinopathy of Coats.

Tuberculosis of the choroid, particularly the conglomerate type, may extend indefinitely, especially in children. The sclera becomes involved and perforation takes place, usually near a vortex vein or an anterior perforating ciliary vessel, a fungating mass appearing under the conjunctiva. Both types in very young children may simulate retinoblastoma, constituting one form of pseudo-glioma (*q.v.*).

The Mantoux dermal reaction is generally used for diagnostic purposes. A positive result is of little value except in children but, of course, does not prove that the ocular condition is tuberculous ; but a negative result makes the diagnosis of allergic tuberculosis unlikely.

Treatment. Streptomycin combined with PAS or Isoniazid (p. 141) is often of great value. In the more chronic and recurrent forms of an allergic nature, improvement frequently follows the use of tuberculin injections, but they should be used cautiously, commencing with very small doses which should be very gradually increased, since a violent reaction may have a serious effect upon the eye. In all cases the usual constitutional treatment should be strictly enforced. In isolated conglomerate tuberculosis of the choroid diathermy or light-coagulation, as used in detachment of the retina (*q.v.*), may be employed to destroy the lesion. If, however, the eye is extensively involved and does not respond to specific treatment, it is best to enucleate it and thus remove a dangerous focus from which the organism may be disseminated into the system.

Brucellosis (*Undulant Fever ; Malta Fever ; Melitensis*). Infection by *Brucella* (*abortus*, *suis* or *melitensis*) is widespread throughout the world and among the many sites of its manifestations the eye may be affected. Initially there is an acute phase of generalized systemic infection ; this is followed by a chronic phase characterized by intermittent bouts of low fever, late in which the ocular manifestations occur. Keratitis and optic neuritis are rare ; a uveitis of a chronic granulomatous nature is more common (Fig. 188). The disease is prone

to relapse and diagnosis can only be suggested from other forms of chronic iridocyclitis or choroiditis by an agglutination test, a cutaneous test, or an opsonocytophagic test. Treatment, apart from the usual measures, is by the sulphonamides or Aureomycin; vaccines have also been employed but, in general, treatment is unsatisfactory.

Toxoplasmosis a protozoan infection derived mainly from rodents and birds, affects both the retina and choroid to form a chorioretinitis; it is probable that in infants the primary seat is most commonly the retina which is involved in association with the brain, while in adults the uveal tract is a frequent primary site. For convenience both types of infection will be discussed here.

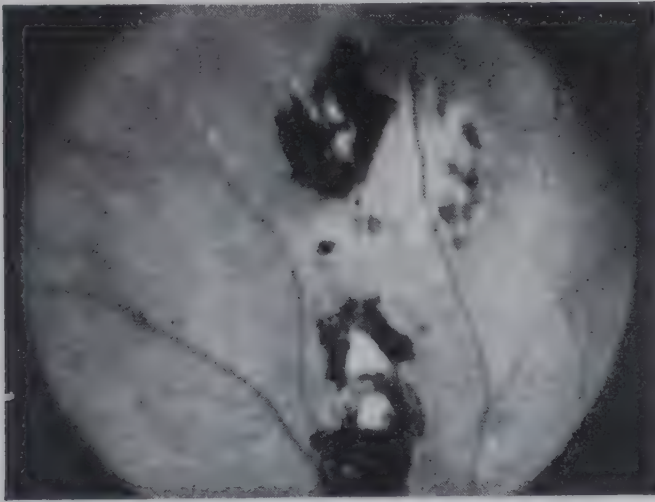


FIG. 196. Toxoplasmosis. Recurrent lesion alongside old scars (Perkins).

In infants, in whom the infection frequently occurs in foetal life, the ocular lesion is usually associated with an encephalitis, but although almost every tissue of the body may be affected, the retinal picture is so common and often so characteristic as to suggest the diagnosis. In the fundus there are bilateral and frequently multiple chorioretinal lesions, the macular area being particularly involved. The whole thickness of the retina and choroid is destroyed in a necrotizing inflammation so that a punched out, heavily pigmented scar remains, clinically indistinguishable from that left by a tuberculous lesion; it is probable that many cases labelled congenital "colobomata" due to intra-uterine inflammation have this ætiology (p. 260). This lesion is frequently associated with well-marked meningeal changes of the same type. Such infants are usually acutely ill with a history of convulsions and, if they survive, may show hydrocephalus, areas of calcification in the brain, and mental retardation.

In adults toxoplasmic infection is rare in the anterior segment of the uveal tract but probably constitutes one of the more common causes of choroiditis (in Europe). The lesion is usually widespread and is characterized by severe recurrent attacks associated with massive exudation into the vitreous; after many relapses it may result in serious visual impairment.

Pathologically the lesion shows an appearance very similar to a tuberculous infection, the characteristic feature being a wide area of necrosis of the retina wherein the parasites may be found, either free or encysted (Fig. 196). Apart from the demonstration of the parasite, diagnosis depends on serological tests (the dye test and the complement-fixation test). Treatment is difficult; pyrimethamine (Daraprim, 25 mg. per day) gives the most satisfactory results.

Sarcoidosis (Benign Lymphogranuloma). Boeck's sarcoid is frequently complicated by a granulomatous iridocyclitis which in many respects resembles tuberculosis. The inflammation is often severe and chronic and is typically characterized by the appearance of multiple nodules which gradually increase in size on the surface of the iris, in the crypts and on the pupillary margin (Koeppel's nodules), but a non-descript type of iridocyclitis may occur. Keratic precipitates are usually gross and of the "mutton-fat" variety. The diagnosis is made from the presence of other systemic manifestations of the disease such as pulmonary changes and areas of rarefaction in the bones. The course of the iridocyclitis is chronic and the prognosis poor.

Uveoparotitis (Heerfordt's disease) is a bilateral affection characterized by a simultaneous involvement of the entire uveal tract, the parotid gland and frequently of the cranial nerves. It appears in young people between ten and thirty years of age, commencing with malaise and fever, sometimes accompanied by a skin rash resembling erythema nodosum. Approximately half the cases commence with a granulomatous iridocyclitis with nodules on the iris, half with a painful swelling of the parotid resembling that due to mumps; subsequently diplopia due to palsy of the ocular motor nerves or facial paralysis is prone to occur. The disease is self-limiting although the iridocyclitis may cause permanent visual damage; the parotid swellings last for six weeks to two years but also ultimately subside. The condition is probably a manifestation of sarcoidosis.

Recurrent iridocyclitis with hypopyon (Behçet's syndrome) is a serious condition wherein a severe iridocyclitis usually characterized by a hypopyon is associated with evidences of a generalized infection accompanied by ulcerative lesions in the conjunctival, oral and genital mucosæ, together with neurological and articular manifestations. It is seen particularly in young adults, and recurs periodically and persistently in attacks of extreme severity so that eventually the vision is usually seriously impaired or lost. The infection is probably viral, but no specific treatment is known, so that routine non-specific measures only are available.

Closely related to this clinical syndrome are two other conditions often included with it under the term *erythema multiforme (exudativum)*. Both of them involve systemic disturbances and widespread mucosal complications—

Reiter's disease (q.v.) and *Stevens-Johnson's disease* (q.v.). In the first, the uveitis is usually more benign and a hypopyon is more rare. In the second, the mucosal infections are predominant and the intra-ocular complications still more rare.

Uveitis associated with vitiligo, poliosis and deafness (the Vogt-Koyanagi syndrome) is a rare condition occurring in young adults. The iridocyclitis is chronic and is associated with an exudative choroiditis which often leads to an exudative detachment of the retina. The ocular inflammation is accompanied by a patchy depigmentation of the skin and the hair, the eyebrows and lashes also whitening (poliosis). *Harada's disease* is a closely related condition affecting mainly the choroid and complicated by an exudative detachment of the retina. The cause is unknown but may be viral.

Heterochromic Iridocyclitis of Fuchs. This is a low-grade chronic cyclitis the only apparent features of which are a lightening of the colour of the affected iris and the presence of a few keratic precipitates on the cornea: the latter distinguish the condition from congenital heterochromia (p. 259). The iris becomes atrophic, loses its markings and readily transilluminates in circumscribed areas, and a cataract frequently develops. The condition is usually said to be associated with some disturbance of the sympathetic nerve supply; this nerve controls the chromatophores accounting for the depigmentation, as well as the tone of the blood-vessels, so that, in their dilated condition, white cells escape and become deposited on the cornea as precipitates. The cataract has a good operative prognosis, but secondary glaucoma may develop due possibly to vasomotor changes.

Sympathetic Ophthalmitis, see p. 400.

DEGENERATIVE CHANGES IN THE UVEAL TRACT

Degenerative Changes in the Iris. Depigmentation of the iris with atrophy of the stroma is seen in old people and, indeed, is a constant senile phenomenon. Depigmentation of the pupillary margin is common and may occur in the form of small triangular patches or radial fissures. Irregular lacunæ in the pigmentary epithelium may often be seen with transillumination, either with the slit-lamp or by contact illumination.

Essential (progressive) atrophy of the iris is a disease of unknown ætiology characterized by a slowly progressive atrophic change in the tissues of the iris which leads to the complete disappearance of large portions of this tissue. The disease usually starts insidiously in early adult life by the development of large areas of atrophy which coalesce and progress to the formation of lacunæ. Vision is eventually lost by the gradual onset of glaucoma due to shrinkage of the tissues at the angle of the anterior chamber. The prognosis is poor; but fortunately the disease is usually unilateral.

Iridoschisis is a rare condition which occurs most commonly as a degenerative senile phenomenon although it may follow as a late result of severe trauma. Large dehiscences appear on the anterior mesodermal layer of the iris and strands of this tissue may float into the anterior chamber as if teased out by a needle; occasionally extensive areas of this layer may become detached. Treatment is unsatisfactory.

Degenerative Changes in the Choroid. Degenerative conditions are more frequent and more important in the posterior than the anterior part of the uveal tract. They may be post-inflammatory or primary.

Secondary degenerations following inflammatory lesions culminating in localized spots of complete atrophy have already been considered. The loss of nourishment to the retina causes atrophy of the outer layers and migration of pigment from the pigment epithelium into the more superficial parts of the retina. The pigment tends to become deposited in the perivascular spaces of the veins, so that the retinal veins may be mapped out here and there by pigment. More noticeable ophthalmoscopically are jet-black branched spots of pigment resembling bone corpuscles, and standing out in sharp relief, an appearance seen in its most typical form in pigmentary retinal dystrophy (*q.v.*). An almost identical picture, although usually without the characteristic distribution of the pigmented spots, may result from choroidal atrophy due to other causes such as syphilis.

Primary choroidal degenerations may be localized or general: the localized forms are usually central although circumpapillary changes around the disc are not infrequent in myopia (*q.v.*) or the late stages of glaucoma.

CENTRAL CHOROIDAL ATROPHY is most commonly the result of myopia or obliterative vasosclerosis, essentially an age change.

Myopic choroido-retinal degeneration. We have already seen that in pathological axial myopia degenerative changes are common, particularly marked around the optic disc and in the central area of the fundus involving the choroid and retina. These changes are not due to the mechanical effects of stretching but are primary in nature and in their incidence genetic factors play a prominent part. They have been erroneously described as "myopic choroiditis" but the condition is not inflammatory. They do not run parallel to the degree of myopia and tend to occur after mid-adult life whereas the elongation of the eye is a phenomenon characteristic of the latter part of the first and the second decades. They probably involve both the ectodermal (retinal) and mesodermal (choroidal and scleral) tissues; but since from the clinical point of view the atrophic changes in the retina are secondary to those in the choroid, the entire clinical picture will be considered here. This includes atrophic changes in the sclera, around the disc, in the choroid and retina in the central area and in the peripheral parts of the retina.

In the majority of cases of moderate myopia there is a *myopic crescent* (Plate X, Fig. 2). This is a white crescent at the temporal border of the disc; very rarely it is nasal. In higher degrees of myopia it may extend to the upper and lower borders, or a complete ring may be formed around the disc.

PLATE X



FIG. 1. Myopic degeneration.



FIG. 2. Myopic retraction and crescent.



FIG. 3. Glaucomatous cup.

[To face p. 254.]

CHOROIDAL ATROPHIES



FIG. 1. Central areolar choroidal atrophy.



FIG. 2. Central guttate choroidal atrophy.



FIG. 3. Senile macular degeneration.

The cause of the myopic crescent has given rise to much discussion ; it may be absent in high myopia and is often present in low, and is essentially atrophic and not merely due to stretching. It is probably congenital in origin, allied to other congenital crescents (p. 351). Anatomically there is considerable distortion of the disc ; on the temporal side the pigment epithelium stops short at a variable distance from the disc and here the choroid is atrophic (Figs. 197, 198). In well marked cases the retina, including the pigment epithelium, encroaches over the nasal edge of the disc (supertraction crescent).

The atrophic thinning of the sclera is confined to its posterior half in which region this structure may be very attenuated.

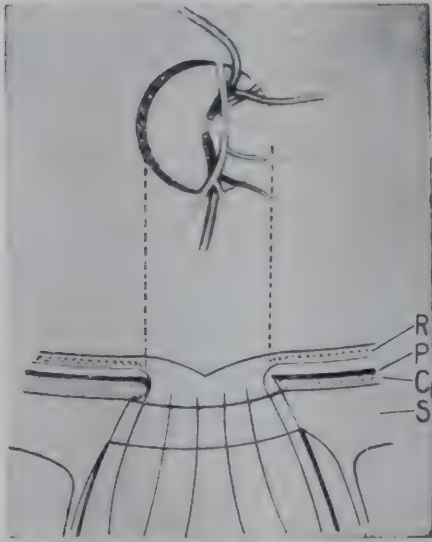


FIG. 197.

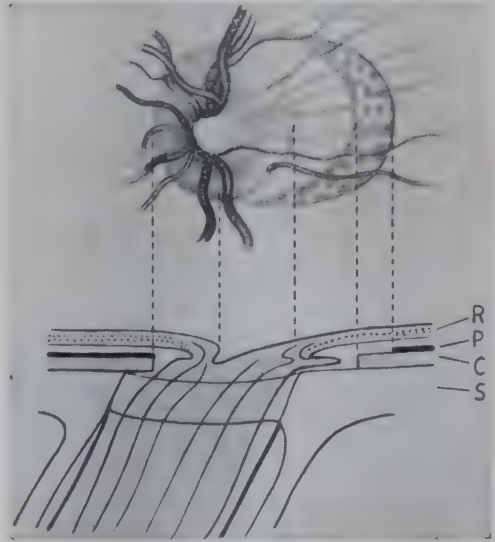


FIG. 198.

FIG. 197 shows the normal configuration of the optic disc; Fig. 198, the myopic. R, retina. P, pigmentary epithelium. C, choroid. S, sclera. On the temporal side the choroidal and scleral crescents are delineated. Note the oblique temporal direction of the optic nerve fibres and the overlapping on the nasal side resulting in supertraction.

Atrophic changes in the choroid (*myopic choroido-retinal atrophy*) occur mainly in the central area of the fundus (Plate X, Fig. 1). There is a gradual disappearance of the small vessels of the choroid with the development of lacunæ making irregular areas of atrophy which may extend to the region of the disc where they may eventually fuse with each other and with the myopic crescent so as to form an irregular circumpapillary ring. Small hæmorrhages are not uncommon in the macular area and occasionally choroidal thromboses ; the latter may give rise to the sudden formation of a circular claret-coloured or black spot at the fovea which may persist (*Fuchs's fleck*). These changes are associated with an atrophy of the overlying retina and involve considerable loss of visual acuity ; this

tends to be progressive and a central scotoma may result. At the same time the retinal pigmentary epithelium becomes depigmented over most of the fundus so that the choroidal vessels are well seen.

Degenerative changes at the periphery of the retina are also common, typically those of cystoid degeneration (*q.v.*). These may lead to the formation of retinal holes resulting in a detachment of this tissue. Degenerative changes also occur in the vitreous which turns fluid with a breakdown of its colloid structure so that dusty opacities or large membrane-like "floaters" are formed.

These atrophic changes are one of the commonest causes of grave visual disability; treatment is ineffective.

Senile central choroidal atrophy assumes two chief forms. In central guttate choroidal atrophy (Tay's "choroiditis") there are

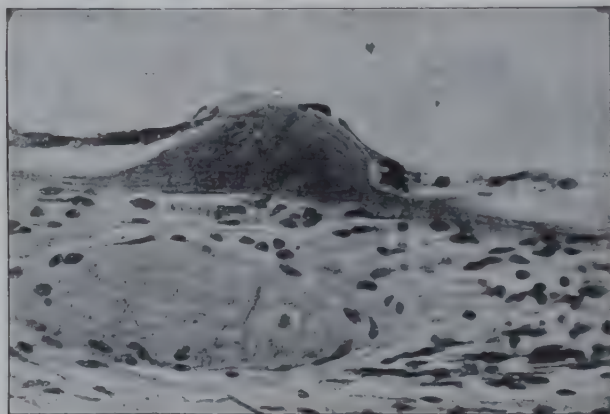


FIG. 199. Colloid body, appearing as a simple sessile bulging of Bruch's membrane and covered by a single layer of flattened epithelium (H. & E. $\times 200$) (Coats).

numerous minute yellowish white spots in the macular region (Plate XI, Fig. 2). They are usually round, but the larger spots may have crenated edges, thus showing signs of fusion. There may be indefinite signs of greyish pigmentation of their edges due to the fact that the pigmentary epithelium is stretched over them. The spots are due to peculiar hyaline excrescences on the surface of the choroid, commonly known as *colloid bodies* (Fig. 199); they are of the same nature as Bruch's membrane, and like it are secreted by the pigmented epithelial cells. The condition is bilateral but causes little impairment of vision, although other senile changes are often present which may cause defective sight.

Central areolar choroidal atrophy (sclerosis) appears as a large circular or oval patch of degeneration in the macular region in which the choroidal vessels are visible owing to atrophy of the retinal pigment epithelium (Plate XI, Fig. 1). As a result of the atrophy of the choroid the sclera shines through and the patch appears white, although traversed by choroidal vessels. Only the larger choroidal vessels are

seen, the smaller ones having disappeared, and even the large ones may appear smaller than usual owing to degeneration of their walls when they may appear densely white and sclerotic. There is an absolute central scotoma. Occasionally the degeneration progresses slowly to involve most of the fundus. This form of central atrophy is genetically determined.

Senile Macular Degeneration. Much more commonly, minute changes limited to the area at and immediately around the fovea occur in old people and lead to grave disturbance or abolition of central vision (Plate XI, Fig. 3). It is generally necessary to dilate the pupil in order that they may be seen. In the early stage the fovea is surrounded by a ring of very fine pigment spots. The stippling is more sharply defined on the foveal side, which usually has a circular or crenated edge, and diminishes rapidly peripherally, where the fundus becomes normal. The fovea gradually becomes paler in colour and the stippling denser, the change being associated with gradually increasing failure of vision, until eventually the small central scotoma becomes absolute. Both eyes are affected, but one is usually attacked before the other which may not be involved for many months. In the majority of cases the atrophy is due to an obliterative sclerosis of the small vessels in the subjacent choriocapillaris.

Disciform Degeneration at the Macula (Junius-Kuhnt's Disease). Sometimes a round white or yellowish patch, about the size of the disc or larger, is seen at the macula and may be bilateral (Fig. 200). Frequently it appears as a raised mass of fibrous tissue. The patient is usually old with obvious disease of the retinal vessels but, on the other hand, these may appear relatively normal. The lesion is the result of obliteration of the choroidal vessels with the proliferation of fibrous tissue following repeated hæmorrhages which burst through Bruch's membrane.

Essential (Gyrate) atrophy of the choroid is a condition of unknown ætiology characterized by progressive atrophy of the choroid, the pigment epithelium and the retina, starting usually with patchy distribution in early adult life, at first in irregular areas which coalesce so that in the final stages practically all the fundus has disappeared, with preservation only of the macula.

Choroideremia resembles the terminal stage of gyrate atrophy. The condition is a hereditary degeneration and the most prominent symptoms are night-blindness and extreme concentric contraction of the visual fields.

Treatment. The treatment of these degenerative changes is very unsatisfactory. When central senile changes are seen early the treatment of any source of debility or vascular disease should be carried out and the general health improved in every way. Particular attention should be paid to the cardio-vascular system so that the circulation is maintained through the small vessels; a mild vasodilator such as nicotinic acid (50 to 100 mg. t.i.d.) may be

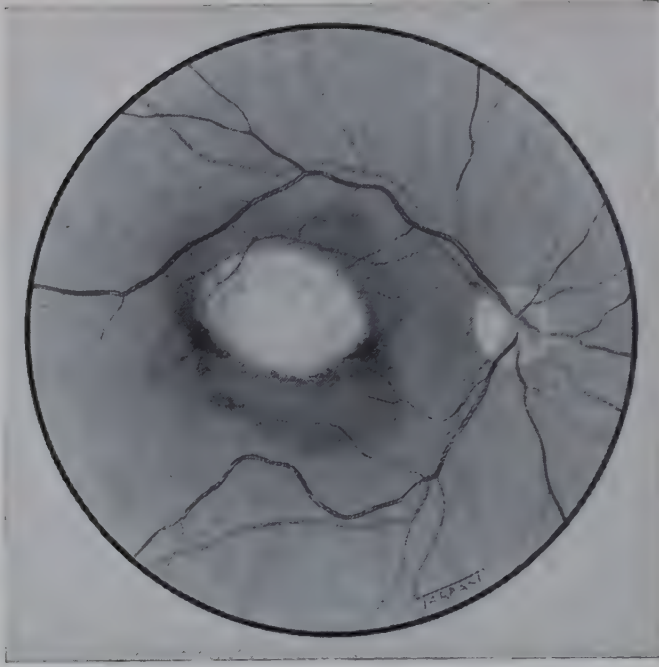


FIG. 200. Disciform Degeneration of the Macula. The central fibrous mass is surrounded by hæmorrhage.

tried but is rarely effective. The patient should be assured that peripheral vision will not become affected, so that, although unable to read or do fine work, he will always be able to see sufficiently well to get about freely. In the early stages reading is facilitated by the use of a magnifying reading aid (p. 81).

Detachment of the Choroid. The choroid is often apparently detached from the sclera in eyes which have been lost by plastic iridocyclitis or glaucoma, and this may also result from severe hæmorrhage or a new growth. These cases cannot be diagnosed clinically. The condition also not uncommonly occurs during the first days following intra-ocular operations owing to the great vasodilatation and exudation into the outer lamellæ of the choroid which follow the sudden lowering of the intra-ocular pressure. The anterior chamber is shallow and on ophthalmoscopic examination the detached choroid is seen through the pupil as a dark mass; it may also be visible as a dark brown mass by oblique illumination. The prognosis is usually good, the choroid becoming replaced and the anterior chamber re-established. Occasionally, however, the tension of the eye remains low, and if the iris remains long in contact with the cornea at the angle of the anterior chamber, peripheral synechiæ may form so that an obstructive glaucoma may eventually develop. In such circumstances it is well to ensure that the wound or the conjunctival flap is not leaking; if it is it should be reconstituted, if necessary by a conjunctival flap from above. In recalcitrant cases

in which the choroidal detachment shows no evidence of subsiding, drainage of the subchoroidal fluid through a diathermy puncture at the most prominent region of the detachment has been suggested while the administration of acetazolamide has sometimes proved useful.

CONGENITAL ABNORMALITIES OF THE UVEAL TRACT

One iris may have a different colour from the other (*heterochromia iridum*), or parts of the same iris, usually a sector, may differ in colour from the remainder (*heterochromia iridis*). The blue iris is due to the absence of pigment in the iris stroma, the pigment in the retinal epithelium being seen through the translucent stroma.

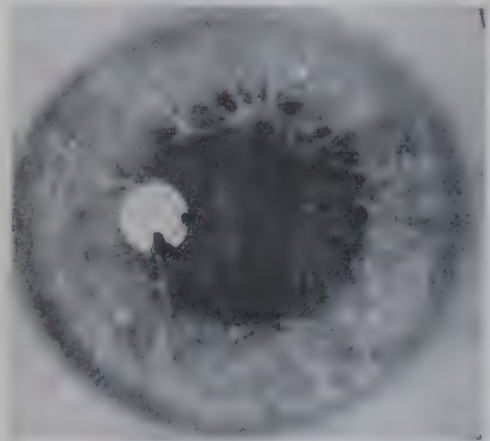
The iris often shows patches of brown pigmentation; these *benign melanomata* are due to abnormal groups of pigmented cells lying in the posterior layers of the stroma.

The pupil is normally slightly to the inner side of the centre of the cornea. In some cases it is considerably displaced, usually also to the nasal side—*corectopia* (κόρη, pupil, ἐκ, out of, τόπος, place). Rarely there are other holes in the iris besides the pupil—*polycoria*.

The iris may be apparently absent—*aniridia* or *irideremia*—a condition which is usually bilateral; there is, however, a narrow rim persistent at the ciliary border, but it is hidden from view during life by the sclera. On examination the ciliary processes and the suspensory ligament of the lens can be seen. There is a tendency for secondary glaucoma to develop due to the abnormal structure of the angle of the anterior chamber.

Persistent pupillary membrane is due to the continued existence of part of the anterior vascular sheath of the lens, a foetal structure which normally disappears shortly before birth. Fine threads stretch across the pupil, or may be anchored down to the lens capsule (Fig. 201). They are distinguished from post-inflammatory synechiæ in always coming from the anterior surface of the iris just

FIG. 201. Persistent pupillary membrane.



outside the pupillary margin, from the position of the circulus iridis minor. Such tags are of frequent occurrence and are of no pathological importance. They are commonest in babies and probably undergo some absorption as age advances, but many persist permanently. Examination with the slit-lamp shows that minute remnants of the pupillary membrane are very common even in adults.

The foetal pupillary membrane consists of a network of small blood vessels supported by a very delicate stroma which contains pigment cells. Sometimes the pigment is left on the lens surface and persists. It forms a stippling of very fine brown dots scattered over a circular area of 5 or 6 mm. in diameter in the centre of the pupil. These spots are distinguished from the pigment spots left by posterior synechiæ which have broken down (p. 230) in being much smaller, stellate in shape when magnified under the slit-lamp, much more numerous and regularly arranged, and also by the absence of any concomitant signs of iritis. They do not usually interfere with vision.

Colobomata. Colobomata form one of the commonest congenital malformations of the eye (but are nevertheless rare) in which the tissues of the uvea and the associated retinal tissues or their prolongation onto the back of the iris are badly developed, or deficient.



FIG. 202. Coloboma of the iris.

As a rule they are due to defective closure of the embryonic cleft (p. 1) in which case they occur in the lower part of the eye (*typical colobomata*). A *coloboma of the iris* may involve this tissue only, when it is usually pear-shaped, the deficiency extending from the pupil towards, but not always as far as, the ciliary body, usually running downwards and slightly inwards (Fig. 202). Colobomata of the iris found in other directions are called *atypical*. A coloboma of the iris may be associated with a similar *coloboma of the choroid and retina*, or the latter condition may occur alone.

A typical coloboma of the fundus appears as an oval or comet-shaped defect with the rounded apex towards the disc, which may or may not be included (Plate XVI, Fig. 3). A few vessels are seen over the surface, some retinal, others derived from the choroid at the edges. The surface is often depressed irregularly (ectatic coloboma). The central vision is generally bad, and there is a scotoma in the field corresponding more or less accurately to the coloboma, although this usually contains some retinal elements near the edges.

Similar patches, often symmetrical in the two eyes, occur in other situations (*atypical coloboma*), notably at the macula (*central or macular coloboma*). It is probable that some of these are due to intra-uterine inflammation, particularly toxoplasmosis (*q.v.*).

Albinism is a hereditary condition in which there is a defective development of pigment throughout the body. Owing to absence of pigment in the eye the iris looks pink and the patients suffer from dazzling. Nystagmus, photophobia, and defective vision are usually present and occasionally strabismus. With the ophthalmoscope the retinal and choroidal vessels are seen with great clarity, separated by glistening white spaces where the sclera shines through (Plate III, Fig. 3). Microscopic examination has shown that total albinism is extremely rare, as traces of pigment have always been found in the retinal epithelium.

Partial albinism is commoner, wherein the absence of pigment is limited to the choroid and retina, the irides being blue. The macular regions are pigmented and may therefore look normal. People with dark hair sometimes have relatively slight pigmentation in the periphery of the fundus so that the choroidal vessels are seen : these patients will be found to have had fair hair as children.

Treatment consists in the use of tinted glasses as a protection from glare.

Cysts of the Iris. *Serous cysts* of the iris sometimes occur and are due to closure of iris crypts with the retention of fluid. *Cysts of the posterior epithelium* occur, due to accumulation of fluid between the two layers of retinal epithelium. They look like an iris bombé limited to parts of the circumference—a limitation which is impossible in the case of true iris bombé (*q.v.*). In these cases the posterior layer of epithelium is often adherent to the lens. *Implantation of epithelium* into the iris sometimes occurs after perforating wounds or operations, giving rise to pearl cysts. When such wounds heal badly the corneal epithelium may occasionally spread over the iris and line the whole anterior chamber, causing glaucoma. Such cases are not true implantation cysts. Eyelashes are sometimes carried into the anterior chamber in perforating wounds and, lodging upon the iris, may be associated with cysts formed by the proliferation of the epithelium of their root-sheaths.

Tumours of the Uveal Tract. See Chapter 25.

CHAPTER 19

THE LENS

BEING composed entirely of epithelium surrounded by a capsule, the lens is incapable of becoming inflamed. Some of its fibres, however, are frequently poorly developed and degenerative changes occurring during life are common. Either of these conditions leads to a loss of transparency in the parts affected. As a general rule, developmental opacities are partial and stationary, acquired opacities progressive until the entire lens is involved. Any opacity in the lens or its capsule whether developmental or acquired, is called *cataract*.

Care, however, should be taken in using this term clinically since it often excites anxiety which may be entirely unjustified. This applies particularly to the stationary types of opacity ; and it is to be remembered that even in senile cataract the opacities may remain localized for years without causing serious disability, or sometimes without being suspected. It is often wise (and kind), therefore, to tell such patients that they have "lens opacities" and, if pressed, to suggest that the development of cataract may be long delayed and can be dealt with adequately should the need ever arise.

DEVELOPMENTAL CATARACT

Developmental cataract assumes the most variegated forms and is common in its minor manifestations ; indeed, the lenses of most people show minute points of opacity of this type when examined with the beam of the slit-lamp under full mydriasis. We have seen (p. 9) that the lens is formed in layers, the central nucleus being the earliest formation, around which concentric zones are subsequently laid down, the process continuing until late adolescence (Fig. 12). Developmental cataract has therefore a tendency to affect the particular zone which was being formed when this process was disturbed ; the fibres laid down previously and subsequently are often normally formed and remain clear. As time goes on, such an opacity is thus usually deeply buried in the substance of the lens by the subsequent formation of normal fibres. Developmental cataract thus tends to follow the architectural pattern of the lens and from its location an estimate can be made of the stage of development at which the anomaly occurred.

What the deleterious influences may be which may cause such developmental anomalies are yet largely unknown. Maternal (and infantile) malnutrition is certainly one, as in zonular cataract ; maternal infections by viruses another, as in rubella ; deficient

oxygenation owing to placental hæmorrhages possibly a third. Such cataracts tend to be stationary, although progressive opacification of a senile type may occur earlier than is usual. From the functional point of view, most of them are of little or no significance unless they are considerable in size and central in position.

Among the many morphological types the following are the most common.

Punctate cataract is the most common manifestation and in minute degree is almost universal in occurrence. When the small opaque spots are multiple and scattered all over the lens, appearing as tiny blue dots by oblique illumination with the slit-lamp, they are



FIG. 203. Anterior axial embryonic cataract.

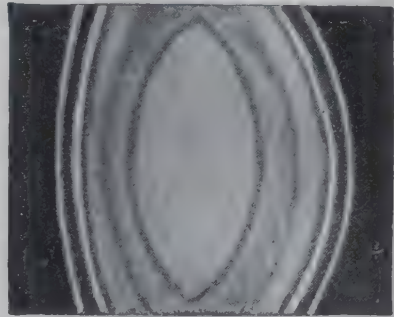


FIG. 204. Embryonal nuclear cataract.

known as *cataracta cœrulea*, or *blue-dot cataract*; when crowded in the Y-sutures, the terms *sutural cataract* and *anterior axial embryonic cataract* (Fig. 203) have been applied. None of these is of significance.

Fusiform cataract, also called *spindle-shaped*, *axial*, or *coralliform*, is an antero-posterior spindle-shaped opacity, sometimes with offshoots giving an appearance much resembling coral (Fig. 206): it is genetically determined and shows a tendency to occur in families. *Discoid cataract* is also a familial form, showing a somewhat ill-defined disc of opacity just behind the nucleus in the posterior cortex.

When the development of the lens has been inhibited at a very early stage, the central nucleus remains opaque—(*embryonal*) *nuclear cataract* (Fig. 204). Ordinarily this is of little significance.

A progressive type of congenital cataract, originally nuclear, is associated with the occurrence of *rubella* (German measles) in the mother if the infection is contracted in the second and sometimes in the third month of pregnancy. It is presumed that the virus damages the lens at this early stage. Pathologically the nucleus is found to be necrotic and the whole lens becomes opaque. Other congenital anomalies occur in association with the cataract, particularly congenital heart disease (patent ductus arteriosus), microphthalmos, micrencephaly, mental retardation, deafness and dental anomalies. The frequency of this combination with maternal rubella contracted about the second month of pregnancy raises the question of the advisability of exposing to infection intending mothers who have not had the disease, or the more

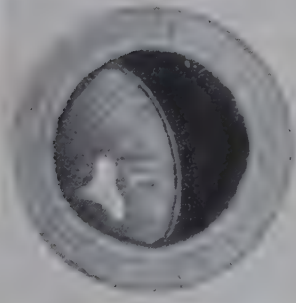


FIG. 205. Ectopia lentis with a capsular cataract.

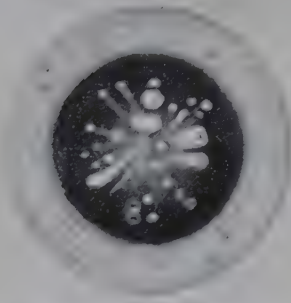


FIG. 206. Coralliform cataract.



FIG. 207. Zonular cataract.

serious question of abortion if such an illness develops at this crucial stage in pregnancy. The possibility of other viruses traversing the placental barrier is still a matter of conjecture.

In **zonular cataract**, development has been interfered with at a later stage and an area around the embryonic nucleus becomes opacified, its extent depending on the duration of the inhibiting factor. The opacity is usually sharply demarcated and the area of the lens within and around the opaque zone is clear, although linear opacities like spokes of a wheel (called *riders*) may run outwards towards the equator. Occasionally two such rings of opacity are seen. Such cataracts are usually bilateral and if, as is frequently the case, they are formed just before or shortly after birth they may be of sufficient diameter to fill the pupillary aperture when the pupil is undilated ; they may thus affect vision (Fig. 207).

Such zonular cataracts may have a genetic origin with a strong hereditary tendency of the dominant type. On the other hand, they may be due to a period of malnutrition at some stage of late intra-uterine or early infantile life. Lack of vitamin D is apparently a potent factor and other evidences of rickets are commonly found in affected children. This deficiency inhibits the development of other epithelial structures, particularly the enamel of the permanent teeth which is being formed at the time ; the permanent incisors and canines particularly have an eroded appearance with transverse lines across them.

Coronary cataract represents the same type of developmental cataract occurring about puberty ; it is therefore situated in the deep layers of the cortex and the most superficial layers of the nucleus. It appears as a corona of club-shaped opacities near the periphery of the lens, usually hidden by the iris, while the axial region and the extreme periphery of the lens remain free (Figs. 208 and 209). The opacities are not progressive and do not lead to complete opacification of the lens : their importance lies in their

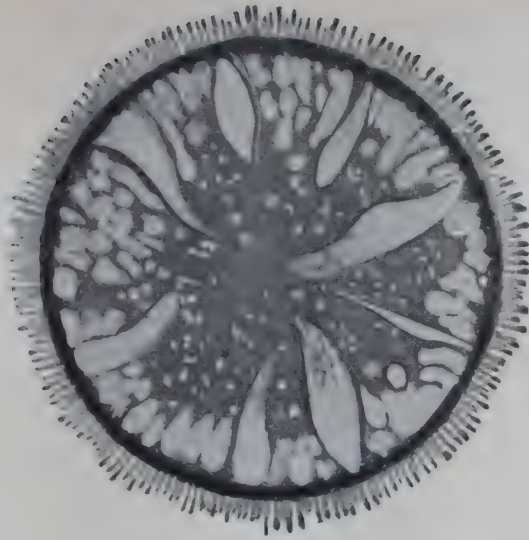


FIG. 208.

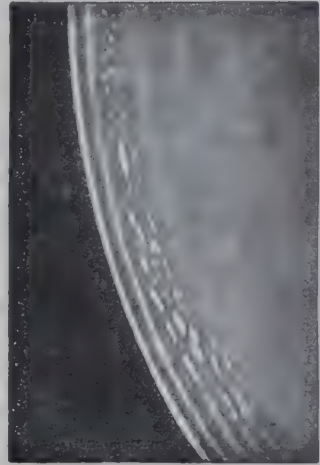


FIG. 209.

FIGS. 208-209. Coronary cataract by oblique illumination (Fig. 208) and in optical section with the beam of the slit-lamp (Fig. 209).

recognition as a developmental anomaly, for if they are seen when the pupil is dilated and their character is not recognized, the examiner may be led to diagnose a progressive cataract in a young adult.

Anterior capsular (polar) cataract may be developmental owing to delayed formation of the anterior chamber; in this case the opacity is congenital. More commonly the condition is acquired, and follows contact of the lens capsule with the cornea, usually after the perforation of an ulcer in ophthalmia neonatorum (*q.v.*). Fortunately such a reaction is seen only in early infancy. Where contact has occurred, usually in the central pupillary area, a white plaque forms in the lens capsule (Fig. 205); sometimes it projects forwards into the anterior chamber like a pyramid (*anterior pyramidal cataract*) (Fig. 210). Occasionally the underlying layers of cortex are opaque forming an *anterior cortical cataract*. When this occurs it may well be that the subcapsular epithelium grows in between the capsular and cortical opacities so that the clear lens fibres subsequently

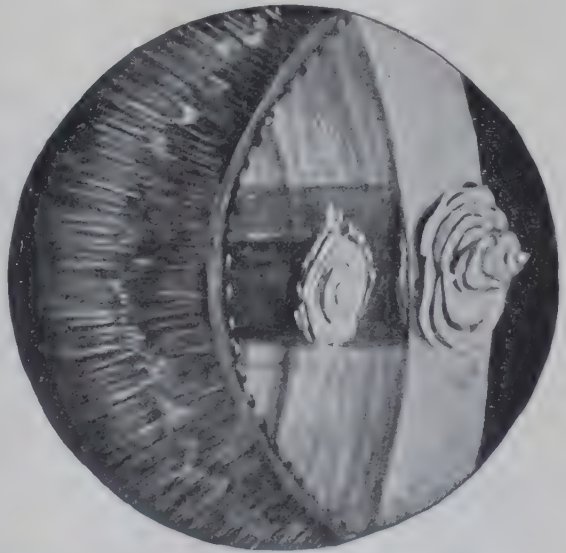


FIG. 210. Anterior polar cataract with an imprint (reduplicated cataract). (Harrison Butler).

growing therefrom lay down a transparent zone between the two opacities. The buried opacity is called an "*imprint*" and the two together constitute a *reduplicated cataract*. Such opacities are not progressive and rarely interfere with vision.

Posterior capsular cataract is due to persistence of the posterior part of the vascular sheath of the lens. In minimal degrees it is common and usually insignificant. Sometimes, however, particularly in cases of persistent hyaloid artery, the lens is deeply invaded by fibrous tissue and a *total cataract* is formed (Fig. 229) (p. 284).

Treatment is not required in developmental cataract unless vision is considerably impaired. If the cataract is central and reasonably good vision can be obtained through clear cortex around it, the child should be kept under mydriasis with atropine at least until puberty. An optical iridectomy is an alternative measure if the opacity is stationary. If the opacity is large, the operation of *discission (needling)* (Chapter 27) may be undertaken whereby the capsule is perforated; in children the entire lens is absorbed if the aqueous gains access to it. The operation, however, should not be performed unless the vision is seriously impaired. A decision on this question depends upon whether vision with corrected refraction and retained accommodation is to be preferred to probably improved vision after operation without accommodation. Vision of 6/12 or even 6/18, with retained accommodation is probably more valuable than a problematic 6/9 without accommodation, to which must be added the disability of constantly wearing very strong convex glasses for distance and still stronger ones for near work. Contact lenses, however, can often be worn by relatively young children or can be made available in later life. In such eyes, however, the vision is frequently subnormal.

It is not advisable to needle lamellar cataracts until the child is one or preferably three years old, but when the lens is completely opaque, or the pupil will not dilate, and when squint or nystagmus is developing, it may be wise to needle at an earlier age, although there is some risk in doing so. Moreover, in unilateral cataracts in children it is probably better to operate in youth rather than wait for an extraction in adult life, although there is some danger from the eventual development of a retinal detachment. The treatment of unilateral cataract in children thus differs from that of a similar condition in adults.

Discission, however, may be difficult or impossible in total or shrunken cataract, and it may be necessary to remove a lens of this type with capsule forceps (Fig. 308) through a keratome incision. Vitreous is usually lost and there may be a severe reaction, but the result not infrequently justifies the heroic measures. The difficulties may be greater if a persistent hyaloid artery is present. In such cases violent attempts to remove the opacity by needling will result

in the loss of the eye ; the best that often can be done is to displace the opacity from the centre of the pupil.

ACQUIRED CATARACT

Acquired cataract is due to the degeneration of lens fibres already formed. The reasons for the degeneration are not yet clear and probably vary in different cases ; any factor, physical or chemical, which disturbs the critical intra- and extra-cellular equilibrium of water and electrolytes or deranges the colloid system within the fibres tends to bring about opacification.

Biochemically two factors are evident in this process. In the early stages of cataract, particularly the rapidly developing forms, hydration is a prominent feature so that frequently actual droplets of fluid gather under the capsule forming lacunæ between the fibres, and the entire tissue swells and becomes opaque (*intumescence*). To some extent this process may be reversible and opacities thus formed may clear up. Hydration may be due to osmotic changes within the lens or to changes in the semi-permeability of the capsule. The process is seen dramatically in traumatic cataract when the capsule is ruptured and the lens fibres swell and bulge out into the anterior chamber (*q.v.*). If, however, the proteins are denatured a dense opacity is produced, a process which is irreversible ; opacities thus constituted do not clear up.

Such an alteration occurs typically in the young lens or the cortex of the adult where metabolism is relatively active. In the older and inactive fibres of the nucleus it is rarely seen. Here the usual degenerative change is rather one of slow sclerosis. Clinically, when the first process is predominant the condition is called a "soft cataract" ; the second is described as a "hard cataract."

The cause of these changes is obscure. Presumably they depend essentially on interference with metabolism, which, as we have seen, is essentially autoxidative (p. 23). It may be significant that in a fully developed cataract the principal agencies which mediate these autoxidative systems—glutathione, ascorbic acid, riboflavine—are in deficit or absent. It is not known whether the loss of these substances is causal or consequential, but it is certain that a cataractous lens is an asphyxiated lens.

Many factors lead to these changes. The most common is *age* ; and it may be of significance that as age progresses the semi-permeability of the capsule is impaired, the inactive insoluble proteins increase, and the oxidation system is less effective. Experimentally, cataract can be produced in conditions of *deficiency*, either of amino acids (tryptophane) or vitamin B₂ (riboflavine), or by the administration of *toxic substances* (naphthalene, lactose, galactose, thallium, etc.). Dinitrophenol, used for slimming, and paradichlorobenzene, used as an insecticide, also produce lens opacities in the posterior cortex, as also do toxic products in the aqueous (as in cyclitis) (*complicated cataract*). *Hypocalcæmia* may lead to the same result perhaps by altering the ionic balance ; this experimental finding is correlated with the cataract of parathyroid tetany. Cataractous changes may also follow the use of the stronger anticholinesterase group of *miotics* and after the prolonged systemic use

of *corticosteroids*. Physical factors may also enter into the question—*osmotic influences* (as may be largely responsible for diabetic cataract), *mechanical trauma* (traumatic cataract), or *radiant energy* in any form.

Senile Cataract is rare in persons under fifty years of age unless associated with some metabolic disturbance as diabetes, but is almost universal in some degree in persons over seventy. It occurs equally in men and women and is usually bilateral, but often develops earlier in one eye than the other. There is a considerable genetic influence in its incidence and in hereditary cases it may appear at

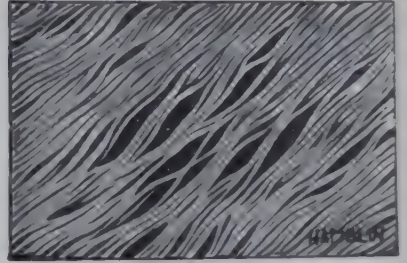


FIG. 211. Lamellar separation.



FIG. 212. Fluid vacuoles under the capsule and in the cortex (seen by the slit-lamp).

an earlier age in successive generations.

Two types of senile cataract may occur—cortical cataract, wherein the classical signs of hydration followed by coagulation of proteins appear primarily in the cortex, and nuclear or sclerotic cataract wherein the essential feature is a slow sclerosis in the nucleus.

In the *cortical type of senile cataract*, *pre-senile changes* are the rule, the most characteristic of which is a demarcation of the cortical fibres owing to their separation by fluid. This phenomenon (*lamellar separation*) can only be seen with the slit-lamp and is invisible ophthalmoscopically (Figs. 211–12). The general increase in the refractive index of the cortex in old people gives a grey appearance to the pupil in contradistinction to the blackness seen in the young; the greyness is due, not to cataractous changes, but mainly to the increase in reflection and scattering of light.

In the next stage of *incipient cataract* wedge-shaped spokes of opacity with clear areas between them appear in the periphery of the lens (Fig. 213); they lie in the cortex, some in front of and some behind the nucleus (*lens striæ*), and are preceded by sectorial alterations in the refractive indices of the lens fibres thus producing irregularities in refraction, some visual deterioration and polyopia. The bases of the wedge-shaped opacities are peripheral and they are most common in the lower nasal quadrant. At first they can only be seen with the pupil dilated, but as they develop their apices appear within the normal pupillary margin. With oblique illumination they appear grey; seen with the ophthalmoscope or mirror they are black against the red background of the fundus; and as they approach the axial area, vision becomes seriously disturbed (Plate IV, Fig. 2).

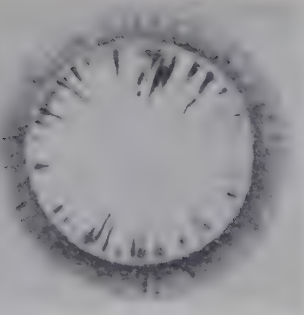


FIG. 213. Early cataract showing lens striæ.

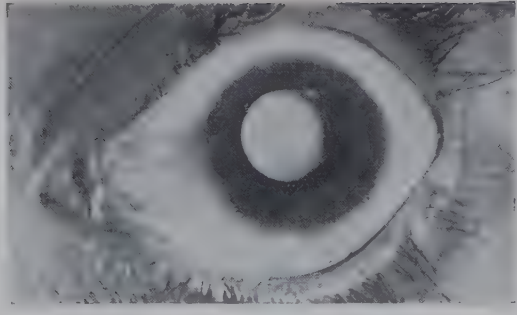


FIG. 214. Mature cataract.

As time goes on, opacification becomes more diffuse and irregular so that the deeper layers of the cortex become cloudy and eventually uniformly white and opaque. Meanwhile the progressive hydration of the cortical layers may cause a swelling of the lens, thus making the anterior chamber shallow (*intumescent cataract*). Eventually the entire cortex becomes opaque, the swelling subsides and the cataract is said to be ripe or *mature* (Fig. 214). In the meantime the nucleus suffers little change except a progressive sclerosis.

As long as there is any clear lens substance between the pupillary margin of the iris and the opacity, the iris throws a shadow upon the grey opacity when light is cast upon the eye from one side (Fig. 215). When the cortex is completely opaque the pupillary margin lies almost in contact with the opacity, separated only by the capsule; the iris then throws no shadow, and the cataract is known to be mature (Fig. 216). This used to be an important guide to the most favourable time for operation by the extracapsular method.

If the process is allowed to go on uninterruptedly the stage of *hypermaturity* sets in when the cortex becomes disintegrated and

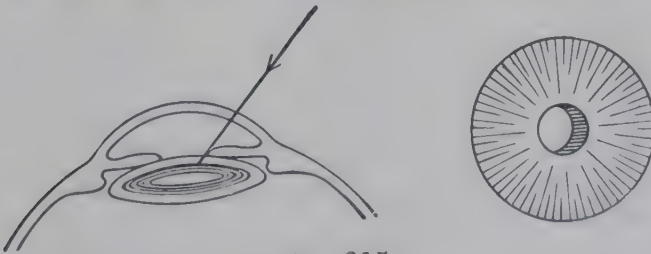


FIG. 215.

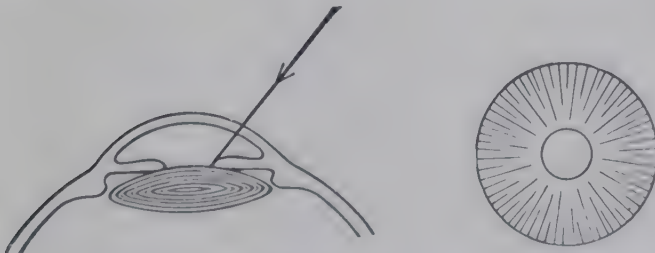


FIG. 216.

FIGS. 215-216. Diagnosis of the maturity of a cataract.
(The pupil is illuminated from the right of the figure.)

transformed into a pultaceous mass. The lens becomes more and more inspissated and shrunken, sometimes yellow in appearance, often with cretaceous deposits and bright specks due to crystals of cholesterol. The anterior capsule becomes thickened by proliferation of the anterior cubical cells, so that a dense white capsular cataract is formed at the anterior pole in the pupillary area. Owing to shrinkage the lens and iris become tremulous and the anterior chamber deep, and finally, degeneration of the suspensory ligament may lead to luxation of the lens.

Sometimes at the stage of maturity the cortex becomes fluid, and the nucleus may sink to the bottom of the lens. The liquefied cortex is milky, the nucleus appearing as a brown mass limited above by a semi-circular line, altering its position with changes in position of the head. Such a cataract is called a *Morgagnian cataract* (Fig. 217).

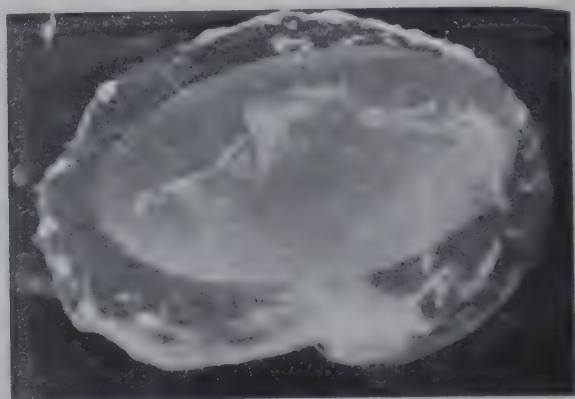


FIG. 217. Morgagnian cataract.

The rate of development of senile cortical cataract varies greatly, sometimes occupying many years; indeed, the cataract may never reach maturity. Very rapid maturation in younger patients usually indicates some complication such as cyclitis or diabetes. The forms with fine radial lines evolve more slowly than those with cloudy opacities. It is best to examine every case periodically, a careful drawing of the opacities being recorded at each visit.

A second common type of cortical senile cataract is *cupuliform cataract*, consisting of a dense aggregation of opacities just beneath the capsule, usually in the posterior cortex. It is difficult to see with the ophthalmoscope but appears in the beam of the slit-lamp as a yellow layer. Examination with this instrument is important since, being near the nodal point of the eye, the opacity may diminish the vision considerably in older people.

In *senile nuclear sclerosis of the lens* the opposite process occurs; the normal tendency of the central nuclear fibres to become sclerosed is intensified while the cortical fibres remain transparent.

This type of cataract tends to occur earlier than the cortical variety, often soon after forty years of age. As time progresses the nucleus becomes diffusely cloudy, the cloudiness spreading gradually towards the cortex, and occasionally it becomes tinted dark brown, dusky red or even black owing to the deposition of melanins derived from the amino acids in the lens (*cataracta brunescens*; *black cataract*) (Fig. 218). In maturity the sclerosis may extend almost to the capsule so that the entire lens functions as a nucleus. Such cataracts require a large corneal section for their removal.

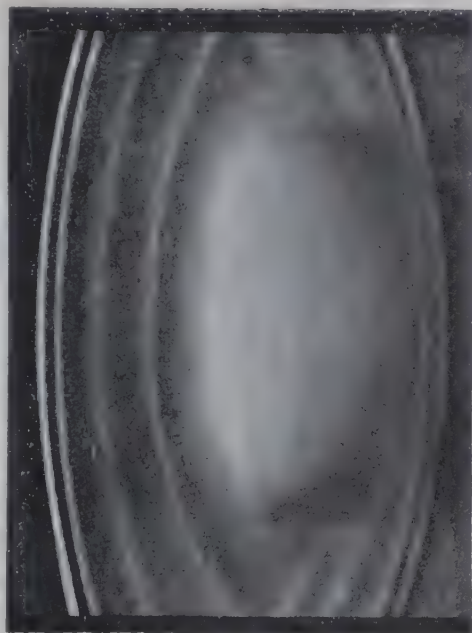


FIG. 218. Senile nuclear sclerosis.

Initially little change may be seen with the ophthalmoscope except that the details of the fundus are hazy. Occasionally if there is much pigment the pupillary reflex may be entirely blackened. There is, however, considerable visual disturbance—at first a progressive myopia owing to the increased refractive index of the nucleus, and then general impairment of vision; but progress is usually very slow and hypermaturity does not occur.

Cataract associated with Ocular Disease

Complicated Cataract results from a disturbance of the nutrition of the lens due to inflammatory or degenerative disease of other parts of the eye, such as iridocyclitis, choroiditis, high myopia, pigmentary retinal dystrophy or retinal detachment. After inflammations of the anterior segment, a nondescript opacification appears throughout the cortex which usually progresses and matures rapidly. In inflammations or degenerations affecting the posterior segment a characteristic opacification usually commences in the posterior part of the cortex in the axial region (*posterior cortical cataract*). Ophthalmoscopically it appears as a vaguely defined, dark area, and with the slit-lamp the opacity is seen to have irregular borders extending diffusely towards the equator and the nucleus (Fig. 219); unlike developmental cataract it is not sharply confined to a particular zone (Fig. 220). In the beam of the slit-lamp the opacities have an appearance like bread-crumbs and a characteristic rainbow display of colours often replaces the normal achromatic sheen (*polychromatic lustre*). Such a cataract may remain stationary in the posterior cortex for a long time or even indefinitely; in other

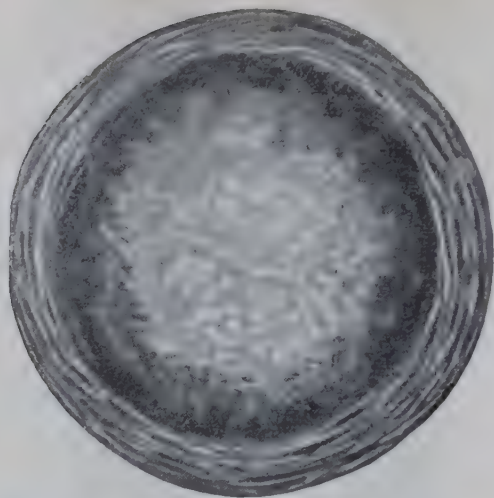


FIG. 219. Posterior cortical cataract.



FIG. 220. Optical section of the posterior pole in the beam of the slit-lamp, showing the position of the opacity.

cases the opacification spreads peripherally until all the posterior cortex is affected, and axially until the entire lens is involved. The total cataract formed in this manner is usually soft and uniform in appearance. In the later stages the capsule becomes thickened, the whole lens shrinks giving rise to tremulousness of the iris, and other degenerative changes such as calcification ensue.

Even in the early stages vision is usually much impaired owing to the position of the opacity near the nodal point of the eye. The operative prognosis depends on the causal condition; but the presence of such a cataract without obvious cause should always call for a most minute examination of the eye for keratic precipitates or other signs of disease.

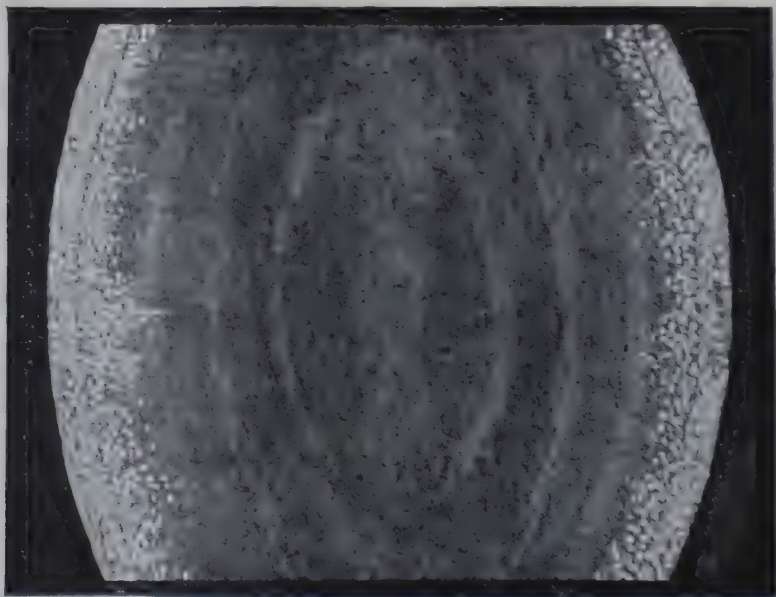


FIG. 221. Diabetic cataract. Optical section as seen with the slit-lamp. (Goulden.)

Cataract associated with Systemic Disease

Diabetic Cataract. We have seen that in diabetic subjects senile cataract tends to develop at an earlier age and more rapidly than usual. *True diabetic cataract* is a rare condition occurring typically in young people in whom the diabetes is so acute as to disturb grossly the water-balance of the body. An immense number of fluid vacuoles appears under the anterior and posterior parts of the capsule, producing a diffuse opacity which at this stage is reversible. Thereafter a cloud of opacities resembling snow-flakes appears all over the cortex until the lens rapidly assumes a uniform milky-white colour (Fig. 221). The operative prognosis depends on the possibility of controlling the general disease.

In **Parathyroid Tetany** cataractous changes may occur when the parathyroid glands become atrophic or have been inadvertently removed in the course of a thyroidectomy; development may be prevented by the administration of parathyroid hormone and calcium. Clouds of small discrete opacities appear in the cortex separated from the capsule by a clear zone (Fig. 222). These coalesce to form large, glistening, crystalline flakes and within six months the lens is usually opaque. The other ocular tissues are unaffected and the operative prognosis is good.

In **Myotonic Dystrophy** a characteristic and somewhat similar cataract may develop. In a sharply limited zone of the cortex underneath the capsule both anteriorly and posteriorly, fine dust-like opacities appear interspersed with tiny iridescent spots. The cataract may remain stationary; if it progresses the operative prognosis is good.

Galactosæmia is a rare congenital disease characterized by an inborn inability of the infant to metabolize galactose. It is frequently associated with the development of bilateral cataract in early life, at first lamellar and eventually total. Recession may occur if milk and milk-products are eliminated from the diet in the early stages; otherwise if the patient survives, surgical treatment must be adopted.

Mongolian Idiocy and **Cretinism** may be associated with similar punctate subcapsular cataracts.

An **Atopic Cataract** which tends to develop rapidly, appears frequently in sufferers from severe and widespread skin diseases—atopic eczema, poikiloderma vasculare atrophicans, scleroderma, keratosis follicularis, and others. The operative prognosis depends on adequate control of the allergic condition in eczematous cases.



FIG. 222. Cataract in aparathyroidea.

Cataract due to Radiant or other Energy

Most types of radiant energy produce cataractous changes, particularly heat, X-rays and the γ -rays of radium or neutrons. Ultra-violet light has been implicated as a factor in the ætiology of senile cataract, a suggestion due largely to the common occurrence of this condition in tropical countries such as India. It is true that radiation with massive doses can produce lens opacities in experimental animals, perhaps by inducing changes in the permeability of the lens capsule; but that sunlight has much clinical effect in the production of cataract is problematical.

Heat (infra-red) Cataract is a characteristic condition which may be induced experimentally in animals and occurs clinically in industry. The heat acts not directly on the lens but is absorbed by the pigment of the iris and ciliary body and thus influences the fibres of the lens indirectly; it has thus been found impossible to produce such cataracts experimentally in lightly pigmented or albino animals. The cataract is characteristic in appearance. In the early stages there is a small disc of opacity in the posterior cortex of the lens, thinner and more sharply defined than the posterior cortical opacity of complicated cataract, but it may extend throughout the cortex in the later stages. In addition, the zonular lamella of the capsule may be exfoliated, sometimes in large sheets which curl up in the pupillary area.

Such a cataract is seen industrially in two particular occupations. It is seen in *glass-workers* who have long been engaged in glass manufacture, particularly beer bottles and plate glass, but not in those who make flint-glass bottles or pressed glass articles since the heat of such furnaces is less. It also occurs in certain *iron-workers*, especially tin-plate millmen and chain-makers.

Irradiation Cataract may be caused by X-rays, γ -rays or neutrons. The characteristic changes appear to be due to the direct action of the rays on the dividing cells and developing fibres of the lens itself. The initial changes are found near the equator shortly after radiation, and the first clinical evidences are apparent in the cortex near the posterior pole only after a period of one or two years when the equatorial cells have migrated posteriorly, where they resemble in appearance those of heat cataract. Maturation of the cataract may occur fairly rapidly. Technicians who have been inadequately protected may be thus affected, or patients treated for malignant conditions near the eye. Such cataracts have also developed in workers in atomic energy plants and occurred among the survivors of the atomic bombs released over Japan in the Second World War.

Electric Cataract may develop rapidly after the passage through the body of a powerful electric current as from a flash of lightning or the short-circuiting of a high-voltage current. The cataract usually starts as punctate, subcapsular opacities and matures rapidly. The effect is due partly to heat, partly to concussion and partly to chemical changes following electrolytic dissociation.

Ultrasonic Radiation similarly induces lens opacities, due to the effects of heat and concussion.

TRAUMATIC CATARACT—either due to concussion or a perforating wound—see pp. 379, 388.

The symptoms of acquired cataract are entirely visual. Among the early complaints is that of seeing spots before the eye, not continually moving as in *muscæ volitantes* (*q.v.*), but stationary, retaining their relative position in the field of vision in different positions of the eye. Uniocular polyopia, another early symptom, is the doubling or trebling of the objects seen with the eye. It is due to irregular refraction by the lens so that several images are formed of each object; coloured halos may also be seen (p. 294). There may also be a change in colour values owing to the absorption of the shorter wave-lengths so that reds are accentuated.

As the opacity extends and becomes denser, the acuity of central vision suffers, the deterioration depending on the density and position of the opacity. If the opacities are peripheral, as in senile cortical cataract, serious visual embarrassment may be long delayed and the vision is improved if the pupil is contracted in bright light. If they are central, visual deterioration appears early, and the patient sees better when the pupil is dilated in dim illumination. Posterior cortical opacities often cause diminution of central vision apparently out of proportion to the amount of opacity observed. When nuclear sclerosis is prominent, the increasing refractivity leads to the development of a progressive myopia. It follows that with senile nuclear sclerosis a previously presbyopic patient may be able to read again without the aid of spectacles; he refers to his "improvement" in vision as "second sight."

As opacification proceeds, vision steadily diminishes until only perception of light remains. In many cases of mature senile cataract fingers can still be counted at a few feet, or at least hand movements discerned. In all cases, however, light should be perceived readily and the direction of its incidence accurately indicated.

The Treatment of Cataract. No medical treatment by drugs or otherwise has been shown to have any significant effect in inducing the disappearance of cataract once opacities have developed. When the cataract is at the early stages of hydration and is due to general disease (as diabetes), control of the causal condition may result in a disappearance of the lens changes; if opacification has occurred, control of the general condition may stay its progress; but once the proteins of the lens have become coagulated, the change is immutable. In senile cataract the progress of opacification may cease spontaneously for many years, or refractive changes may result in temporary improvement of vision. From time to time these events have been seized upon as indicating a therapeutic effect of a particular remedy which may happen to be applied. In this way potassium iodide drops, dionine, subconjunctival injections, hormone treatment especially with parathyroid extract, the admini-

stration of calcium salts, and so on, have been enthusiastically advocated ; but all are useless.

In all cases, however, a careful examination of the patient should be made to exclude any specific or constitutional cause of the disease ; if any is found, it should be treated. This applies particularly to diabetes ; the urine of all such patients should be tested.

In *incipient cataract* the condition of the patient may be much ameliorated during the tedious process of maturation. The refraction, which often changes with considerable rapidity, should be corrected at frequent intervals. There is no reason to restrict the use of the eyes in incipient uncomplicated cataract, but the patient may be much assisted by instructions as to the arrangement of illumination. If the pupillary area is free, brilliant illumination will be found best ; if the opacities are largely central a dull light placed beside and slightly behind the patient's head will give the best result. In these cases, also, dark glasses are usually of great value and comfort when worn out-of-doors, an effect obtained with greater certainty by instilling a very weak mydriatic (atropine $\frac{1}{20}$ per cent. every morning), provided—owing to the risk of glaucoma—the angle of the anterior chamber is not narrow. Finally, in every case of incipient cataract the pupil should be dilated to allow a thorough examination of the fundus at the stage when it can still be seen.

When the cataract has become mature the only effective treatment is its operative removal. Before this is contemplated, however, a *general overhaul* of the patient should exclude the presence of serious systemic disease. A disease such as diabetes does not preclude operation but it should be adequately controlled by an expert before and after surgery.

A *thorough examination of the eye* is also a necessity, a routine particularly vital when the cataract appears to be of the complicated type, but eminently desirable in all cases. The retinal function must then be explored since, if it is defective, operation may be valueless and the patient warned of possible disappointment. In this respect the value of a thorough examination of the fundus in the incipient stage of cataract while the retina and optic disc are still visible is obvious. When it cannot be seen four tests are of value.

The detection of the *projection of light* is of the utmost importance. It is tested as follows in a dark room. The opposite eye is covered securely by the palm of the patient's hand. Light is then shone from the ophthalmoscope into the cataractous eye from various directions, the patient looking straight forwards. He is told to point with his other hand in the direction from which the light seems to come. He ought to do this readily and accurately.

The *macular function* can frequently be assessed by asking the patient to look through an opaque disc perforated with two pin-holes close together behind which a light is held. If he can appreciate the presence of two lights, the central area of the retina is probably good. He should also be asked to look at a distant light through a Maddox rod (p. 469) ; if the red line is continuous and unbroken, macular function is probably good.

An *entoptic view of the retina* will often allow the patient to supply valuable information. If the eyes are closed and the globe is steadily and firmly massaged through the lower lid with the bare lighted bulb of an electric ophthalmoscope or torch, he will see clearly the entire vascular tree of the retina on an orange ground. An intelligent patient will describe any blanks or scotomata ; particular attention should be given to the central area.

A foveal electroretinogram may give useful information of the functioning of this region.

If operation is considered worthwhile, disease in the anterior segment should be excluded. The pupil should react promptly and normally to light and it should dilate readily with mydriatics while the most careful search must be made for precipitates on the back of the cornea. When the cataract is complicated by intra-ocular disease, treatment must be directed in the first place to rendering it quiescent—often a tedious and sometimes an unsatisfactory procedure. Some cases are not suitable for operation, mostly on account of cyclitis or defective projection of light, but even in these, if there is a possibility of success, operation may be undertaken after warning the patient of the doubtful issue, for the loss of such an eye weighs little against a reasonable probability of improved vision. In such cases, also, wherein an inflammatory condition may be expected to flare up, the topical administration of steroids is often of great value in forestalling or controlling a relapse.

If operation is decided upon, the possibility of infective complications should be excluded as far as practicable. Gross focal sepsis, such as abscessed teeth, should if possible be eliminated since they may lead to post-operative complications such as hæmorrhage or iritis. This should be done, however, with care, particularly in old and frail people ; an alternative sometimes advisable is to conduct the operation under the umbrella of a suitable antibiotic. The conjunctival sac should be examined, a swab taken, and any infection cleared up by suitable antibiotic treatment. Finally, the lacrimal sac should receive attention. If regurgitation is found on finger pressure (p. 511) or a mucocele is present, it should be excised or a nasal drainage operation performed, the passage being repeatedly irrigated by a suitable antibiotic (Chapter 14).

The type of operation employed depends on the case. In young patients, the lens can be disposed of by *discission* (p. 423), an operation which may require repetition ; this may be obviated by removing the lens material by suction. Such an operation is usually effective up to thirty years of age. In the second and third decades the degree of intumescence thus induced may be considerable and absorption slow, so that an irritative iridocyclitis or a secondary glaucoma may develop some days after discission. In these cases it is well to wash out the soft lens matter through a keratome incision (*linear extraction*) (p. 425) or to remove it by suction. In older people, the nucleus of the lens is not absorbed in this way and must be extracted. The extraction may be done by opening the capsule,

expressing the nucleus, washing out as much of the cortical substance as possible and leaving the remainder to be absorbed (*extracapsular extraction*) (p. 428). When a complete iridectomy is performed during the operation to assist the removal of the lens the technique is termed *combined extraction*. Extracapsular extraction is followed by a needling of the capsule, a procedure which, in the absence of complications, should always be done in about three weeks even although this membrane is thin, since it will almost certainly thicken in time so that the vision will again fall ; the eye, however, should be quiet and free of inflammation. Alternatively, the entire lens including the capsule can be removed by rupturing the zonule (*intracapsular extraction*) (p. 431).

If the cataract is very immature but vision has become embarrassed, the difficulties of extracapsular extraction are increased since the clear soft cortex is difficult to remove from the eye, tends to the production of iritis and other complications, and leads to the formation of dense *after-cataract*. Immaturity, however, is not an absolute bar to this method, but such cases are generally an indication for the intracapsular technique. With the use of either method, however, it is not necessary to wait for maturity since, apart from earning capacity, many of these patients are old and rely much upon reading for their interest in life. Whenever reading or work becomes difficult an intracapsular extraction can be performed.

The treatment of *unilateral cataract* offers some difficulty when the vision is good in the fellow eye. Little advantage is gained by operating since the difference in refraction between the two eyes after operation will be so great that diplopia will result if the refraction is corrected with spectacles (p. 90), and if uncorrected the large blurred image formed by the eye may be a positive disadvantage. Binocular vision is often possible with a contact lens (*q.v.*), particularly in children, but their manipulation is difficult by the aged ; and the technique of inserting a plastic lens into the eye (*vide infra*) may provide a way out of the difficulty but this procedure is by no means easy. Neglecting these two expedients, the sole advantage gained from the extraction of a unilateral cataract is an increase of the field of vision on the affected side. This may be a matter of great importance, as in people who work among machinery or who have to go about where there is much traffic ; but as a rule this slight advantage does not justify operation unless the lens shows signs of becoming hypermature.

The *presence of increased tension* constitutes an anxiety in operating for cataract. The tension may be raised owing to the swelling of the lens in the incipient stage, in which case an extraction is indicated. On the other hand, simple glaucoma may be already present. If the glaucoma is medically controlled, the lens may be extracted and the treatment continued ; if it is not, probably the best procedure is to perform a combined cataract extraction and a drainage operation such as Scheie's sclerectomy with cauterization.

In some cases eyes with cataract have had surgery for simple glaucoma (p. 307). Theoretically it is obviously objectionable to make a cataract

incision through a drainage area, but although such cases often do well it is probably best to make the incision in the upper part of the cornea in front of the drainage area; an alternative is to make the incision for the cataract extraction in an area separate from the filtering bleb as in the infero-temporal quadrant, but this is technically difficult.

The correction of the refraction after extraction of cataract is dealt with elsewhere (*see, Aphakia*, p. 89).

To obviate the many and obvious disadvantages of aphakia the ingenious suggestion has been made of inserting a small lens of acrylic plastic into the eye as a substitute for the cataractous lens. If this lens is of correct optical power, normal binocular vision with no, or low-powered, correcting lenses is obtained, although, of course, accommodation is lacking. The safest technique is that of Strampelli, whereby in a subsequent operation a plastic lens is inserted into the anterior chamber in front of the iris supported by struts resting in the angle; considerable deformity of the ciliary body, however, tends to result owing to long-continued pressure and the operation should not be undertaken lightheartedly particularly in young people, at any rate until more long-term results have become available.

After- or Secondary Cataract is the opacity which persists or follows after extracapsular extraction or discission of the lens. In both these operations the posterior and part of the anterior capsule remain *in situ*. In many cases these remnants are fine, forming a thin membrane which is difficult to see (Fig. 223). In other cases, especially when

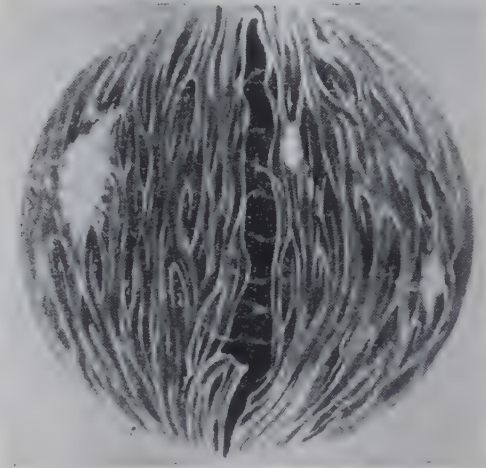


FIG. 223. After-cataract. The thin gossamer membrane is seen with a gaping slit formed by the operation of discission.

the cataract was not mature, some soft, clear cortex sticks to the capsule. This becomes partially absorbed by the action of the aqueous but often becomes shut off by adhesion of the remains of the anterior to the posterior capsule. In such cases the cubical cells which line the anterior capsule also persist; they continue to fulfil their function of forming new lens fibres, although those formed under the abnormal conditions are abortive and opaque. Sometimes these, enclosed between the two layers of capsule, form a dense ring behind the iris (*the ring of Sömmerring*) (Fig. 224); it may cause subsequent trouble by becoming dislocated into the anterior chamber. At other times, the subcapsular cells proliferate and instead of forming lens fibres, develop into large balloon-

like cells which sometimes fill the pupillary aperture (*Elschnig's pearls*). If these remnants lie in the pupillary area a dense membrane is formed so that vision is impaired. If the previous operation has been followed by iritis, exudates also adhere to the lens remnants and organize, thus contributing a fibrous membrane in addition.

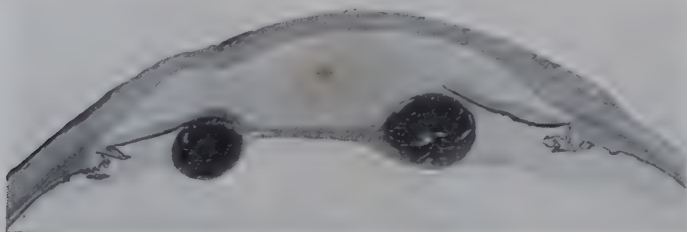


FIG. 224. Sömmerring's ring (Ashton).

Treatment is by discission (p. 423); if proliferation of the capsular epithelium continues, its growth may be inhibited by X-rays.

Dislocation of the Lens. See p. 380.

CONGENITAL ABNORMALITIES OF THE LENS

Besides the various forms of *congenital cataract* (p. 262), abnormalities in the shape and position of the lens occur, often associated with other malformations of the eye.

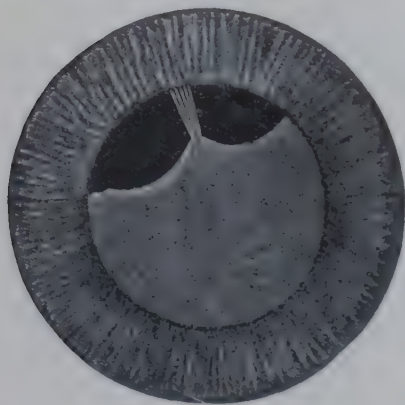


FIG. 225. Coloboma of the lens (Marcus Gunn). The direction of the coloboma, upwards, is somewhat unusual. Note the defective development of the suspensory ligament of the lens.

Coloboma of the lens is the condition in which there is a defect usually in the inferior margin and notch-shaped; less frequently it occurs in some other part of the margin (Fig. 225). It is due to defective development of part of the suspensory ligament.

Ectopia lentis or congenital dislocation is a subluxation of the lens, usually upwards or up and in, and bilateral (Fig. 205). The condition is often hereditary. The lens is small, but the edge is generally invisible until the pupil is dilated. The usual signs of subluxation (p. 380) are then seen. It is sometimes associated with arachnodactyly (*Marfan's syndrome*).

Lenticonus, generally posterior, is an abnormal curvature of the lens, so that

the surface is somewhat conical instead of spherical.

If these deformities cause great visual disability, treatment by discission of the lens is advisable.

CHAPTER 20

DISEASES OF THE VITREOUS

THE vitreous humour is an inert, jelly-like structure which subserves only optical functions. It has all the properties of a hydrophilic gel, undergoing turgescence and deturgescence and readily becoming transformed into a sol when its protein basis becomes coagulated, a transformation which may occur on the slightest insult, either mechanical, chemical or by a change in reaction. Hence "fluid" vitreous is a common condition. It possesses no blood vessels in post-natal life and is incapable of becoming inflamed. We have, therefore, to deal only with symptomatic conditions.

Opacities. Black specks floating before the eyes are commonly seen by normal persons in favourable optical conditions. These *muscæ volitantes* are opacities of various kinds; viewed entoptically, they throw shadows upon the sentient elements of the retina thus appearing as dark spots in the field of vision. Any relatively non-transparent bodies situated anterior to the rods and cones are therefore able to produce muscæ. To this category belong the corpuscles circulating in the retinal blood vessels; if it were not for the fact that the retina is normally adapted for red light the entoptic images of the circulating corpuscles would be a serious impediment to clear vision. Other muscæ are due to minute specks in the vitreous, so small and translucent that they cannot be seen objectively with the ophthalmoscope.

In abnormal conditions vitreous opacities may be so increased as to interfere with vision and to become visible with the ophthalmoscope. They may take the form of threads and flakes, representing the coagulated basis of the gel, as occurs in degenerative conditions as myopia. On the other hand, they may be exogenous and exudative in type, minute albuminous coagula and aggregations of leucocytes, indicating some disease of the uveal tract, particularly cyclitis and retino-choroiditis. In their slightest manifestation they are dust-like opacities, which may permeate the whole vitreous or be limited to the anterior part so that a plane mirror and magnification by a convex lens are necessary in order to distinguish them (p. 124) (Fig. 227). Larger opacities are found after hæmorrhage into the vitreous. They float about, showing that the vitreous is fluid. Vision is often best in the morning, before the muddy vitreous has been stirred up by movements of the eyes.

Dense vitreous opacities obscure the view of the fundus with the ophthalmoscope. In moderate cases the disc and vessels may be

made out, as if seen through a dense haze, in which event the disc may look redder than usual suggesting the appearance of papillitis (*q.v.*). Very frequently in the slighter cases no objective signs of disease can be made out in the fundus ; the foci are either too fine to be appreciated or are anterior to the field of ophthalmoscopic vision in the periphery of the choroid or in the ciliary body.

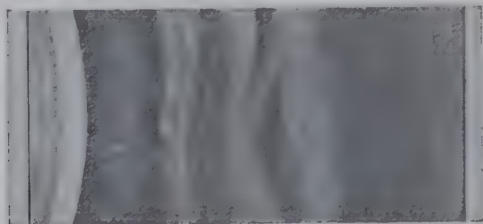


FIG. 226. The normal vitreous seen with the slit-lamp. The posterior surface of the lens is to the left, behind which is a relatively optically empty space occupied by primary vitreous. Posteriorly dense membranous-like masses of gel are seen.

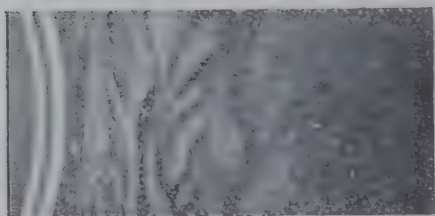


FIG. 227. The vitreous in iridocyclitis showing punctate and membranous opacities.

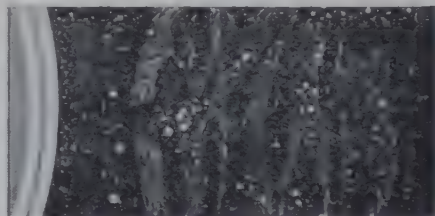


FIG. 228. The vitreous in synchysis scintillans.

Treatment. Slight cases of *muscæ volitantes* without objective signs require no direct treatment. Patients should be advised to ignore the spots as much as possible as they are often only visible when attention is specially directed to them. Treatment of the more severe cases of vitreous opacities depends upon the cause. When this is known, as in iridocyclitis or retino-choroiditis, attention must be directed to the treatment of the primary foci. When the cause cannot be discovered no effective treatment is available. Iodides have been given for their supposed absorptive effect, subconjunctival injections with a view to producing a hyperæmia, and so on ; but the results are disappointing. When all hope of visual recovery has been abandoned, the heroic measure of aspirating the cloudy vitreous through a large-bore syringe and its replacement by vitreous from a cadaver has been tried, but the results cannot be guaranteed.

Fluidity of the Vitreous (*Synchisis*, *συνχέω*, to pour together, disturb) is due to coagulation of the proteins of the vitreous so that the gel structure is disrupted. It is therefore a common feature

in the cases in which opacities are present and is associated with the same causes ; it may also be simply a senile degeneration. The tension of the eye may be normal, but soft eyes nearly always contain fluid vitreous. The condition is diagnosed by the free movement of any opacities seen with the slit-lamp or ophthalmoscope. Such a breakdown occurs most frequently in the posterior region when a detachment of the vitreous may result. In this event the anterior portion of the gel usually remains intact and its movements may cause tears in the retina to which it is attached, leading to small hæmorrhages recognized by the appearance of vitreous opacities or even a retinal detachment.

The degeneration of the vitreous which leads to fluidity may be associated with the formation of a multitude of angular crystals composed mainly of cholesterol which sink to the bottom of the vitreous chamber but are stirred up by movements of the eye. They then appear as a very beautiful shower of golden rain—*synchisis scintillans* (Fig. 228). There is relatively little interference with vision. *Asteroid bodies*, spherical accumulations of calcium soaps, form a somewhat similar appearance in aged eyes with a solid vitreous.

Fluidity of the vitreous requires no treatment in itself, but it is a complication if any intra-ocular operation is contemplated. In such eyes the suspensory ligament of the lens is often weak, so that prolapse of the vitreous or dislocation of the lens may occur. Even when vitreous is lost during the operation useful vision may yet be retained, the gel being replaced by intra-ocular fluid.

Blood in the Vitreous. Hæmorrhage into the vitreous may result from the tugging of vitreous adhesions to the retina, from arterial disease or inflammation of the retina, from contusions or wounds of the eye, in such general diseases as diabetes, pernicious anæmia or malaria, or without apparent cause. A common cause of hæmorrhages, usually severe and recurrent, is periphlebitis retinæ (*q.v.*). Small hæmorrhages are seen ophthalmoscopically and can be accurately watched, although the surrounding vitreous always contains exudates, unless the blood is subhyaloid (p. 310). Larger hæmorrhages, filling the vitreous with blood, are suspected when no fundus reflex can be obtained. It may then be possible to see a red mass behind the lens by oblique illumination.

Absorption of these hæmorrhages usually takes place without organization, owing to the absence of fibroblasts in the vitreous. When organization occurs, as in so-called retinitis proliferans (*q.v.*), it is most marked near the disc, from which membranes and strands stretch forwards. This is due to the presence of mesoblastic tissue containing potential fibroblasts around the central vessels.

Fibrous Tissue in the Vitreous is found after extensive hæmorrhages have occurred, particularly in proliferating diabetic retinopathy or when there is a constitutional basis of tuberculosis or syphilis, and in cases of plastic iridocyclitis it usually takes the

form of membranes stretching across behind the lens. In premature infants fibrous tissue may appear behind the lens associated with a detachment of the retina (*retrolental fibroplasia*, p. 312).

Pus is found in the vitreous only in panophthalmitis (*q.v.*).

Parasites in the Vitreous. *Cysticercus* is very rarely found in the vitreous in England, though it is less uncommon in some other countries. The actual parasite may be seen ophthalmoscopically as a pearly translucent mass sometimes showing peristaltic movements. The treatment—removal—is difficult.

CONGENITAL DEFORMITIES IN THE VITREOUS

Persistent Hyaloid Artery. The hyaloid artery, which in foetal life nourishes the vitreous and supplies blood to the posterior vascular sheath of the lens, becomes obliterated during the sixth and seventh months, and usually disappears completely before birth (p. 1). A remnant, stretching forwards from its origin on the disc, is normal in some animals. Similar remnants are not very rare in man: they



FIG. 229. Encephalo-ophthalmic dysplasia, showing persistent hyaloid artery, the degenerate retina bunched behind the lens, and cataract.

appear as a strand projecting from the disc into the vitreous. There may be membranes veiling the disc from view in such cases. A remnant of the anterior part of the hyaloid artery can often be seen with the slit-lamp in normal eyes as a whitish coiled strand attached to the posterior capsule of the lens 1 to 2 mm. to the nasal side of the posterior pole. Sometimes a larger portion persists, appearing as a circular spot on the back of the lens, constituting a *posterior capsular cataract* (p. 266); at other times the adjacent posterior cortical lens fibres are cataractous. The hyaloid artery may persist in its entirety with the posterior part of the vascular sheath of the lens, a condition usually associated with degeneration, overgrowth and detachment of the retina so that the tissue may accumulate in a mass behind the lens (*persistent hyperplastic primary vitreous* which, when associated with central nervous anomalies, forms the syndrome of *encephalo-ophthalmic dysplasia*) (Fig. 229). Such a condition constitutes one form of pseudo-glioma, and is untreatable.

CHAPTER 21

GLAUCOMA

GLAUCOMA is a symptomatic condition, not a disease *sui generis*. The characteristic physical sign is increased intra-ocular pressure. It will be clear from the description of the mechanism whereby the normal intra-ocular pressure is maintained (p. 18) that its sustained increase in clinical circumstances may be due to an increase in the formation of the aqueous humour, a difficulty in its exit or a raised pressure in the episcleral veins. Of these, the first and last are of little importance; it follows that glaucoma is essentially due to an obstruction to the circulation of the aqueous at the pupil or to its drainage through the angle of the anterior chamber. The importance of embarrassment of the drainage channels through the canal of Schlemm is thus obvious (p. 16). It must be remembered, however, that if they are blocked, some drainage occurs through the uveo-scleral outflow; such an alternative drainage is typically seen in cases of buphthalmos (*q.v.*). These alternative channels, however, are by no means efficient since the pressure remains higher than normal, and they are incapable of dealing with emergencies when sudden changes of pressure occur.

Other factors may cause a rise in the intra-ocular pressure but their influence is relatively small and transitory. Thus a rise in the blood pressure in the capillaries usually associated with states of congestion causes a raised ocular tension owing to the increased volume of the dilated vessels and the increased transudation of fluid into the chambers of the eye; but this is rapidly neutralized by an increase in the drainage of fluid. Similarly, a swollen lens does not cause a rise of pressure in the eye unless it obstructs the angle by pressure, while even the presence of a large intra-ocular tumour does not raise the tension of the eye unless venous obstruction or embarrassment of the drainage angle occurs. Changes in the osmotic pressure of the blood, as we have seen, have little clinical significance in this respect, and it has been shown that the pressure which can be generated by the vitreous by its swelling or turgescence is negligible, amounting only to a few ml. of saline.

It is convenient to divide such clinical states into two classes—(1) primary glaucoma which comprises two separate conditions, simple and closed-angle glaucoma, and (2) secondary glaucoma due to a specific anomaly or disease of the eye.

SECONDARY GLAUCOMA

Secondary glaucoma may show the characteristics either of closed-angle or simple glaucoma. The following are the more common

causes, most of which are fully discussed elsewhere. The treatment of most is that of the primary cause.

(1) *Inflammatory Glaucoma*. *Hypertensive uveitis* (p. 231), wherein the rise of tension is due largely to uveal engorgement and partly to clogging of the drainage channels by the turbid aqueous and inflammatory exudates.

(2) *Post-inflammatory glaucoma*, due to blockage of the circulation of the aqueous either at the pupil by annular posterior synechiæ (iris bombé) or at the angle of the anterior chamber by the formation of peripheral synechiæ or organized exudative deposits (Fig. 185).

(3) *Secondary glaucoma after perforation of the cornea* either by an ulcer or a traumatic or operative wound. The cause in these cases is partly the vascular congestion due to continued irritation, and partly the mechanical blockage of the drainage angle by anterior synechiæ formed by adhesion either of the iris or lens capsule to the wound.

(4) A massive *intra-ocular hæmorrhage* may cause glaucoma, particularly when following a concussion injury. If the ruptured vessel is large the intra-ocular pressure may be suddenly raised to that of the blood pressure. Even when the ruptured vessel becomes sealed, such an accident is usually associated with gross uveal vasodilatation; the presence of the proteins of the plasma in the aqueous and the cellular elements of the blood block the drainage channels. If the hæmorrhage is in the posterior segment of the eye, the lens-iris diaphragm may be pushed forward almost to touch the cornea, further occluding the drainage angle. As a rule in these cases repeated paracenteses is the best treatment (p. 413).

A *hæmorrhagic glaucoma* may follow thrombosis of the central vein of the retina (*q.v.*); it is due to vascular congestion and the organization of tissue at the drainage angle.

(5) *Secondary glaucoma associated with the lens* may arise in two circumstances. If the lens becomes intumescent, either in the rapid development of cataract or after it has been wounded, the swollen lens obliterates the drainage angle by forcing the root of the iris against the sclera. Unless the condition is rapidly relieved by operation (p. 278), extensive peripheral synechiæ involving a permanent rise of tension will result even although the lens is subsequently removed or becomes absorbed. If the lens has been wounded, lens proteins escape into the aqueous, thus aiding the rise of tension by embarrassment of the filtration channels.

A dislocation of the lens acts similarly. If it is partial, the ciliary body is again irritated and a large segment of the angle of the anterior chamber may be compressed or blocked. In complete dislocation into the anterior chamber, the entire angle may be blocked especially if the iris becomes firmly contracted over the posterior surface of the lens.

(6) *An intra-ocular tumour* may cause a secondary glaucoma, not by its increase in volume, but by infiltration of the angle by neoplastic tissue (p. 365).

(7) *Secondary glaucoma* is readily caused by *venous obstruction*, the drainage of aqueous being then impeded (p. 16). This occurs in such conditions as orbital inflammations, large orbital tumours, and carotico-cavernous communications (*q.v.*).

(8) *Obstructive glaucoma* may arise owing to an organic blockage of the angle of the anterior chamber. This may develop in inflammatory or neoplastic processes. It may also occur as a degenerative condition when the corneal endothelium, secreting a hyaline membrane, may spread across the angle. A particularly intractable type of glaucoma results if the epithelium grows through a badly healed corneal wound and spreads round the angle onto the iris (p. 435). Such epithelial ingrowths may follow perforating injuries to the cornea or an operative section as for cataract.

Glaucoma capsulare used to be considered of obstructive origin. Depositions of an exudative material occur on the iris, the ciliary region and on the capsule of the lens. Clinically these appear as flakes on the anterior capsule of the lens which are particularly evident in the peripheral region where it is rubbed upon by the iris ; the axial region is usually free ; they tend to collect in the angle of the anterior chamber and may obstruct the drainage of the aqueous humour. This material which used to be interpreted as an exfoliation of the superficial layers of the lens capsule is evidence of a widespread degenerative change in the anterior uvea, particularly the ciliary region, which itself may be responsible for the glaucomatous process.

Infantile glaucoma (Buphthalmos ; Hydrophthalmos) is usually of a simple obstructive type, due in most cases to a failure in the development of the tissues in the region of the angle of the anterior chamber ; the iris may not completely separate from the cornea (p. 2) so that the angle remains closed by persistent embryonic tissue and the canal of Schlemm may be deficient or absent (Fig. 230). Aqueous veins, for example, may not be present in these cases. Depending on the degree of obstruction the result is a permanent rise in the tension of the eye, but since the circulation of the aqueous is maintained, although at a lower rate, by the anterior ciliary veins and the uveo-scleral outflow, the rise in tension is usually neither marked nor acute. Both eyes are generally affected, and buphthalmos occurs in boys more often than in girls.

Owing to the extensibility of the sclera the eye behaves differently from the adult organ under this increase of pressure. Not only does the lamina cribrosa give way, producing deep cupping (p. 301) but the entire cornea and sclera stretch so that the globe gradually enlarges ; this stretching and expansibility may mask the increased pressure on clinical examination.

In early cases the first sign may be an œdema of the cornea occasionally with the development of corneal opacities. The picture may resemble that of a keratitis accompanied by photophobia and circumcorneal congestion, resembling an inflammatory condition. At a later stage more discrete corneal opacities appear as lines with a double contour due to rupture of Descemet's membrane. As the disease progresses the entire globe stretches, the thinned sclera of the ciliary region becoming bluish in colour, owing to the

uveal pigment showing through. The junction of the cornea and sclera also stretches, so that the cornea is forced forwards and assumes a globular shape resembling keratoglobus (*q.v.*); care should be taken to differentiate between the two conditions. The anterior chamber is therefore extremely deep (Fig. 230). The lens does not participate in the general enlargement; owing to the expansion of the ciliary region the suspensory ligament is stretched so that the lens is flattened and displaced backwards. This removes some support to the iris, which may become tremulous (irido-



FIG. 230. Buphthalmos, illustrating the deep anterior chamber with an anomalous angle, and the globular-shaped cornea.

donesis). As a result of the expansion, the eyes are usually myopic, although much less so than might be anticipated from their length owing to the flattening of the lens and its displacement backwards. Equilibrium may be established with no further loss of vision but in other cases rapid deterioration occurs.

Sometimes in cases wherein the obstruction is incomplete, signs of raised tension may not appear until puberty. This may be called *juvenile glaucoma*. The type of *chronic glaucoma in young adults* which appears about the third or fourth decade and frequently shows hereditary tendencies may have the same basis.

Buphthalmos in relatively early life frequently occurs with more extensive congenital malformations, the most common of which are

neurofibromatosis (p. 502) and the capillary nævus of the face associated with angiomatous conditions of the choroid and the brain (*Sturge-Weber syndrome*, p. 501).

In the *treatment* of buphthalmos, miotics are obviously useless so that operative treatment has to be attempted. The standard drainage operations for simple glaucoma (trephining, iridencleisis, cyclodialysis), have each occasionally given good results, but these are rarely predictable; the operations usually have to be repeated several times and the visual results are often bad. More effective is the operation of *trabeculotomy* (*goniotomy*) wherein a specially constructed knife introduced at the limbus is swept round the angle of the anterior chamber in the opposite segment of the eye under direct gonioscopic observation. It is presumably effective by opening up the draining passages in those cases wherein the condition is due to the blockage of the corneo-iridic angle by persistent embryonic tissue, and may be supplemented by the operation of *gonio-puncture* whereby a puncture is made through the whole thickness of the trabecular region into the subconjunctival space.

PRIMARY GLAUCOMA

Two well-defined types of primary glaucoma exist which differ from each other in the type of patient affected, their clinical course and symptomatology, and in their prognosis and treatment—*closed-angle* and *simple glaucoma*. As a general rule, the first type is characterized by sudden episodic subacute attacks of raised tension, the most notable features of which are a diminution of vision and the subjective appearance of halos caused by corneal œdema. From the less severe of these attacks the eye may seem to recover to a considerable extent, but subsequent episodes tend to involve a permanent raising of the tension (chronic congestive glaucoma) which may result in blindness (absolute glaucoma) or an acute attack may abolish vision. The second type, on the other hand, develops slowly, quietly and insidiously over many years with a characteristic triad of symptoms—raised tension, typical field defects and cupping of the disc—until in the “absolute” stage the eye becomes intensely hard, all vision is lost and the disc develops a deep atrophic cup. It is to be noted that the differentiation between the two types is not based on tension, for an eye showing the picture of simple glaucoma may have a tension much higher than one with acute glaucoma. The differentiation depends on the mechanism of causation. In the closed-angle type the essential lesion is a closure of the angle of the anterior chamber usually associated with a capillary stasis with increased permeability leading to œdema of the tissues of the eye, while in the simple type the angle is clinically wide and open and signs of uveal congestion and œdema are replaced by sclerotic changes.

Closed-angle Glaucoma. This form of glaucoma occurs typically in persons, usually women, in the fifth or sixth decade ; it may, however, occur earlier or later. It is seen particularly in those who are highly strung and anxious in disposition and usually show an instability in their vasomotor reactions.

The type of eye affected is characteristic. It is usually hypermetropic, the anterior chamber is shallow with the lens-iris diaphragm far forward and *its angle narrow* ; the last feature is a constant characteristic. The narrowness of the angle may be due to the smallness of many of these eyes, the configuration of the ciliary body, and often to the relative size of the lens in comparison with the smallness of the eye so that this tissue crowds the root of the iris against the cornea, narrowing the filtration passages. Narrowness of the angle is often a hereditary characteristic but does not become apparent until the fourth or fifth decade, a development typical of advancing years associated to some extent with the growth of the lens.

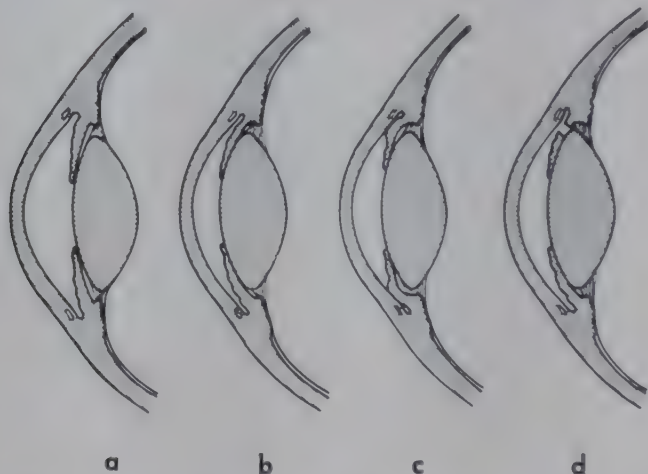


FIG. 231. The anterior chamber and its angle. (a) In a normal eye with a wide angle ; (b) in an eye with a narrow angle ; (c) physiological iris bombé with a semi-dilated pupil ; (d) restoration of the patency of the angle after peripheral iridectomy.

The mechanism of the closure of the angle of the anterior chamber varies. A common sequence of events follows pupillary dilatation. When the anterior chamber is of normal depth, the iris lies flatly in a transverse plane, its pupillary margin touching lightly the anterior surface of the lens (Fig. 231a). In an eye liable to closed-angle glaucoma with its shallow anterior chamber and anteriorly placed lens, the iris is closely apposed to the lens capsule over a wide area with a considerable component of force pressing it against the lens (Fig. 231b) ; communication between the posterior and anterior chambers is therefore difficult so that a condition of *blockage of the pupil* exists. In a state of semi-dilatation of the pupil

the aqueous finds difficulty in passing forwards from the posterior chamber with the result that the relaxed iris bellies forward at its periphery resembling an iris bombé and its root approximates the inner surface of the cornea near the limbus, tending to cut off the filtration channels in the angle (Fig. 231c). In other cases on dilatation of the pupil the iris may become crowded into the angle, or a swelling or anterior displacement of the ciliary body may produce the same effect.

It is possible that in some cases a mechanical occlusion of the angle by the iris is sufficient to block the drainage of the aqueous ; for this reason *the instillation of atropine in an eye with a shallow anterior chamber or a narrow angle is dangerous* since it may precipitate an attack of raised tension. The administration of this drug, incidentally, is without danger in simple glaucoma when the angle is wide. It is to be remembered, however, that by no means every eye with a narrow angle develops glaucomatous attacks ; the association of vasomotor instability, emotional crises and states of fatigue with such attacks suggests that a vascular disturbance of the ciliary body involving œdema and the increased formation of aqueous in the posterior chamber may be an occasional precursor. Since both eyes usually have a similar structure, this type of glaucoma is likely to be bilateral, but one eye is generally affected before the other. If such a sequence of events occurs in an eye with a wide angle, the iris does not approximate the cornea and the drainage channels remain open and can cope with the situation.

The course of the disease may be divided into five stages. In the first or *prodromal stage*, occasional attacks of raised tension occur, characterized by some blurring of vision, the subjective appearance of halos around lights, and occasionally headache. In these attacks the eye remains white, without congestion, even although the tension may suddenly rise for a short period to heights of 40 or 60 mm. Hg. The characteristic symptom is the halos, due to corneal œdema. Such attacks may occur intermittently, being caused by such factors as over-work, anxiety, excitement, fatigue, and particularly by circumstances which cause a pupillary dilatation. At first, however, they are usually transitory and little attention may be paid to them ; occasionally an acute attack may occur at an early stage, but in such cases it is rare not to elicit a history of previous attacks of halos on questioning.

A second *phase of constant instability* is reached when intermittency in these attacks is replaced by regularity. The normal diurnal variation of the ocular tension becomes exaggerated so that it may rise to very considerable heights, particularly in the late afternoon and evening ; rest or sleep frequently induces a spontaneous fall in tension (Fig. 232).

Sooner or later, however, an *acute congestive attack* occurs, always associated with closure of the angle. This differs profoundly from

the prodromal attacks in that the eye, instead of remaining white, becomes suffused and congested, there is acute pain and the tension remains up and, unless relieved by treatment, vision is gravely affected or lost. It is to be noted that although usually high, the tension in such an attack may be considerably lower than that found in the virtually symptomless prodromal attacks; the nature of the two is quite different. The clinical picture suggests that the crisis is due to circulatory strangulation owing to the pressure, perhaps associated with the liberation of histamine-like substances when the rise of tension had been sufficient to cause damage to the tissues.

An acute congestive attack presents a characteristic and dramatic picture. It starts suddenly, intense pain excited by stretching of

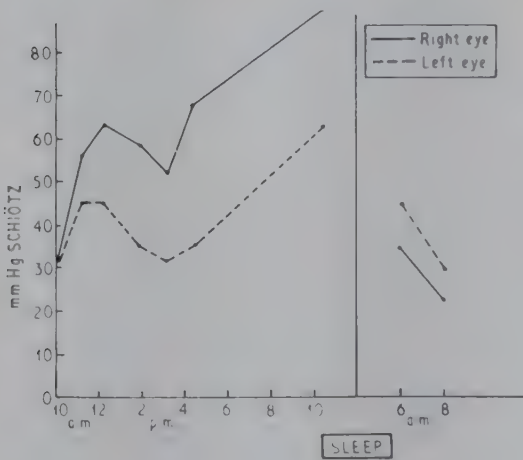


FIG. 232. The diurnal variation of tension in closed-angle glaucoma, showing a tendency to rise progressively late in the evening, and to fall during sleep. The abscissa denotes the time of day; the vertical line indicates when the patient was asleep.

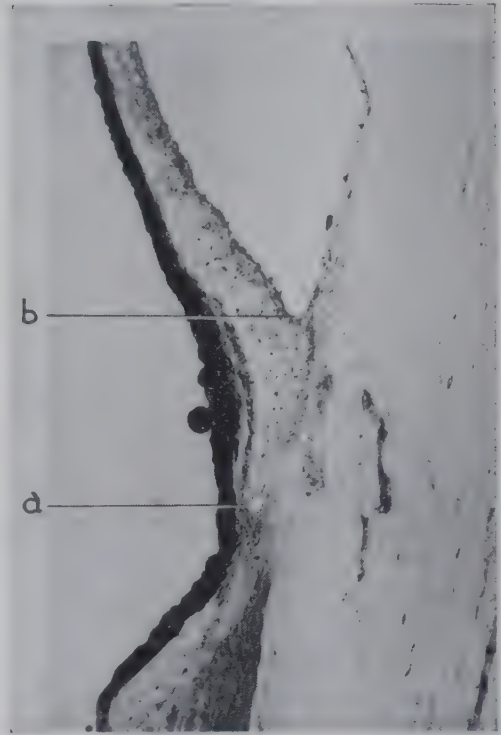
the sensory nerves is felt in the eye and over the entire distribution of the fifth nerve. The pain is frequently so severe that it causes vomiting, and the attack is liable to be mistaken for a severe "bilious attack" wherein the patient is prostrated and the pulse irregular and intermittent. The vision rapidly diminishes, so that in a few hours only hand movements can be recognized. In a considerable number of cases both eyes are affected, an attack in one eye being followed by a similar tragedy in the other.

Objective examination shows some oedema of the lids and conjunctiva; the latter is intensely congested and looks dusky red, owing to the dilatation of the veins. Ciliary congestion is marked; the cornea cloudy and insensitive; the anterior chamber very shallow; the iris discoloured, and the pupil moderately dilated and oval, generally with the long axis vertical. The reactions to light and accommodation are abolished. Ophthalmoscopic examination is impossible owing to the cloudiness of the cornea and the tension of the eye is considerably raised.

If the condition is not relieved by treatment, total abolition of vision may result. Spontaneous improvement, however, may occur,

ushered in by diminution of pain ; but considerable lowering of the visual acuity and, still more, concentric contraction of the field, follow every acute attack. Each attack also tends to produce a permanent adhesion of the root of the iris to the inner surface of the cornea forming *peripheral anterior synechiæ* (Fig. 233). These usually occur earliest and are most numerous in the upper part of the angle but gradually spread around the periphery ; when three-quarters of the circumference has thus been obliterated, the eye reaches a precarious state, for the root of the iris becomes firmly adherent to the back of the cornea forming a “ false angle ” and thus permanently embarrassing drainage. If these adhesions become

FIG. 233. The angle of the anterior chamber in advanced closed-angle glaucoma. (a) The level of the true angle ; the line points to the canal of Schlemm ; (b) the level of the false angle ; (ab) a peripheral anterior synechia ($\times 64$) (Ashton).



sufficiently extensive the tension remains permanently slightly elevated even between attacks, and some congestion and irritability persist. This is the stage of *chronic closed-angle glaucoma*.

Up to the stage of the acute attack the visual acuity remains unimpaired ; after an acute attack it is always depressed. Similarly, before an acute attack the visual fields remain normal ; after an acute attack they become concentrically constricted ; but in the chronic congestive phase the typical scotomatous defects characteristic of simple glaucoma become evident (p. 303). At the same time, cupping of the disc (p. 301) appears for the first time, for the development of this phenomenon in this type of case is a late event depending on prolonged high tension.

The chronic phase, if untreated, with or without the occurrence

of intermittent sub-acute attacks, gradually passes into the final phase of *absolute glaucoma* wherein the eye is completely blind. The anterior ciliary veins are dilated, and a reddish-blue zone surrounds the cornea. The cornea is clear, but insensitive ; it may have vesicles (bullous keratopathy) (Fig. 182) or filaments (filamentary keratitis) upon it. The anterior chamber is very shallow. The iris is dilated, atrophic, and may have a broad zone of pigment around the pupil (ectropion of the uveal pigment). The pupil is grey or greenish, instead of jet black. The optic disc is deeply cupped. The tension is high ; usually the eyeball is as hard as stone. Such an eye is generally painful, with temporary exacerbations, and eventually suffers degenerative changes. The more important are due to a giving way of the sclera before the continued high intra-ocular pressure so that scleral staphylomata are produced ; they may be in the neighbourhood of the ciliary body (ciliary staphylomata) or at the equator (equatorial staphylomata). Such globes may eventually become enormous with walls as thin as paper and there is considerable danger of rupture from slight injury. Sooner or later the tension becomes normal or diminished, due essentially to degeneration of the ciliary body whereby its secretory functions are diminished or abolished ; such an eye may even shrink and suffer widespread degenerative changes.

The *diagnosis* of closed-angle glaucoma in the prodromal stage is of immense importance. In the acute and congestive stages the nature of the condition is usually obvious. The only real difficulty which may arise is its differentiation from iritis ; this has already been discussed (*q.v.*).

In the prodromal stages the diagnosis depends on the history of seeing *halos*, the presence of a narrow angle of the anterior chamber, and the observation or inducement of a rise of tension.

The early diagnosis of closed-angle glaucoma is unusually important since adequate treatment at this stage is easy and as certain of controlling the disease as in any condition in medicine ; after an acute attack has occurred treatment is difficult and its results problematical. *A history of halos, particularly if associated with periodic obscurations of vision, should therefore always excite the liveliest suspicion ; this suspicion should not be diminished by the observation that in the early stages of the disease the eye (apart from its narrow angle) is normal, its tension between attacks is not raised, there is no cupping of the disc and its function (visual acuity and fields) unimpaired, while the facility of the drainage of the aqueous as measured tonographically (*q.v.*) is undiminished.*

The halos are due to the accumulation of fluid in the corneal epithelium and to alterations in the refractive condition of the corneal lamellæ. They are seen as coloured rings around lights and are therefore usually observed after dark. The colours are distributed as in the spectrum with red outside and blue innermost. If the patient gives a

vague history, their appearance can be demonstrated to him by his looking through a thin layer of lycopodium powder enclosed between two glass plates made up as a trial lens.

The only other condition which may commonly give rise to a similar appearance is early cataractous changes in the lens. The two may be differentiated by Fincham's test. A stenopæic slit is passed before the eye across the line of vision. As it passes, a glaucomatous halo remains intact but diminished in intensity, whereas a lenticular halo is broken up into segments which revolve as the slit is moved (Fig. 234).

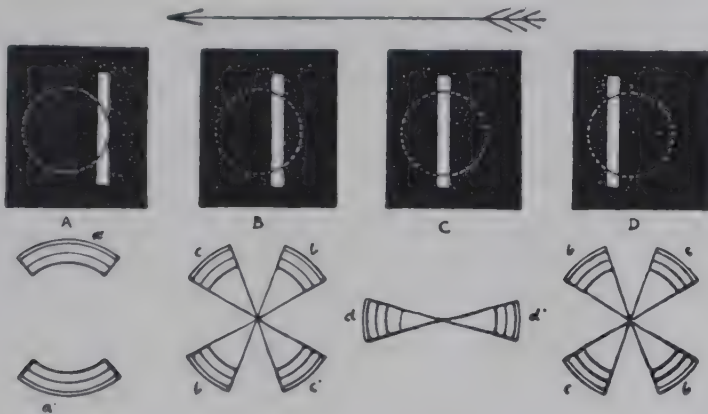


FIG. 234. The stenopæic test. When a stenopæic slit is passed before the eye in the direction of the arrow the successive appearances of the lenticular halo are represented. At A, when the lateral margin only of the pupil is exposed, and the horizontal radial fibres of the lens only are implicated, the horizontal portions *a, a'*, of the halo are visible. At B, when the oblique fibres are involved, the portions *a, a'*, appear to divide, forming *b, c* and *b', c'*; as the slit is moved these rotate, *b, b'* in a clockwise direction, and *c, c'*, anti-clockwise. When C is reached only vertical fibres are involved, and the portions appear to reunite in *d, d'*. On continuing the motion of the slit, the portions *b', c* and *c', b*, appear and revolve similarly, until finally, at the other extreme position, they become amalgamated as *a, a'* in A (Emsley and Fincham).

The anatomical *configuration of the angle* is of immense importance in the diagnosis. Narrowness of the angle is almost invariably accompanied by shallowness of the anterior chamber which itself should give rise to suspicion. This can be seen by ordinary inspection. Examination with the slit-lamp provides a good index of the state of the angle since the degree of approximation of the optical sections of the cornea and iris in the periphery can be readily seen. A narrow periphery, however, may be associated with a wide angle, and the only conclusive evidence is obtained from gonioscopy (*q.v.*) which is invaluable in the diagnosis of the disease. The presence of keratic precipitates or other signs of iridocyclitis should always be excluded by slit-lamp examination to differentiate a primary glaucoma from a hypertensive iridocyclitic crisis (*q.v.*).

The demonstration of a rise in tension is often difficult in the prodromal stages since the hypertensive periods are temporary and often occur at inconvenient times. The patient should be instructed, however, to report to the surgeon whenever he sees a halo ; if this cannot be arranged, *provocative tests* can be undertaken—but always with care in this type of the disease. These are designed to test the control of the ocular tension under conditions of stress.

In the *dark-room test* the patient is placed in a dark room for half an hour ; he must remain awake so that the pupils dilate. Measurement of the tension before and after may show a rise which is pathological if greater than 8 mm. Hg (Schiötz). A better test is to send the patient to a dark cinema to see an exciting film when the factor of emotion is added to that of darkness.

In the *Priscol test* 10 mg. of Priscol are injected subconjunctivally ; a rise of tension greater than 14 mm. Hg (Schiötz) within 60 minutes is pathological.

In the *mydriatic test* the pupil is dilated by a mild mydriatic such as cocaine, neosynephrine or euphthalmine; if these give negative results homatropine may be used, and any rise in tension noted after a period up to two hours. This test should only be used with particular care and only if other evidences of the disease do not exist ; after the test the patient must remain under observation until the surgeon has verified that full miosis is subsequently attained by the instillation of eserine.

If any of these tests are positive—and they are in some 50 per cent. of cases—their evidence is significant. If they are negative, the presence of prodromal acute glaucoma is not excluded, nor is there any assurance that an acute attack will not develop in the immediate future.

The *treatment* of this type of glaucoma should ideally start in the prodromal stage. Whenever it is diagnosed—or suspected—the intermittent attacks of tension should be forestalled by a miotic such as pilocarpine 0.5 to 2.0 per cent. or one of its substitutes. This should be instilled, not necessarily in the morning when the tension in these cases is usually low, but before the halos habitually appear, that is, usually in the late afternoon or evening. The optimum time should be determined in each case by tonometric tests and, in addition, a prophylactic drop should be taken before any periods when fatigue, excitement or darkness is anticipated. If despite this treatment the halos recur—or if the patient is travelling far from adequate surgical care—a prophylactic iridectomy (p. 418) should be performed without hesitation. In early cases before the development of peripheral synechiæ, a peripheral (buttonhole) iridectomy is all that is required ; on its performance, since free communication between the anterior and posterior chambers is now restored, the appearance of iris bombé at once disappears (Fig. 231 d). Such an operation carried out on a quiet eye is practically without risk, involves no visual disability,

and invariably results in a complete cessation of the disease. This is the only type of glaucoma wherein operative treatment can be counted on to be successful and the prognosis is good.

Once an acute attack has developed the outlook is very different. If the case is seen early, every endeavour should be made to lower the tension before operating in order to avoid the difficulties of operation on a congested, chemosed eye, and the dangers of opening a globe the tension of which is high. Full sedation, if necessary with morphia, should be attained. Miotic treatment should be pushed; pilocarpine 1·0 or 2·0 per cent. should be instilled at five-minute intervals for half an hour and then at half-hourly intervals until miosis is secured; the onset of miosis usually coincides with a clearing of the cornea and a fall in tension. The more powerful cholinergic miotics such as DFP are dangerous since they may increase the congestion. At the same time, an attempt may be made to lower the tension by the systemic administration of carbonic anhydrase inhibitors in full doses (Diamox, Neptazane) which may well be supplemented by dehydration measures such as the intravenous injection of urea or mannitol or, more easily and as effectively, by the oral administration of glycerol (1·5 G./kg. body wt.). It is important to realize, however, that these lower the ocular tension by diminishing the formation of aqueous and do not have any effect in opening the angle; even although they lower the tension their use should not be continued for more than a day or so since in these circumstances peripheral anterior synechiæ tend to consolidate and become permanent. Local heat by hot bathings or medical diathermy (p. 564) is frequently of benefit.

A further measure which is sometimes of great value in lowering the tension before operation is the retrobulbar injection of 1 ml. of procaine (4 per cent.) and adrenaline into the region of the ciliary ganglion (p. 562). Not only has this frequently a marked hypotensive effect within a quarter of an hour, but the accompanying anæsthesia relieves the patient's pain and anxiety. It is to be remembered, however, that since paralysis of the ciliary nerves stops the formation of acetylcholine, drugs such as eserine, which act by inhibiting cholinesterase, become ineffective.

If these measures do not show a substantial and rapid effect they should rarely be continued beyond twelve, never beyond twenty-four hours. If the tension still remains high an iridectomy (p. 418) should forthwith be performed. The operation is most conveniently done under basal anæsthesia, and when the eye is congested the systemic administration of sympatholytic drugs such as hexamethonium may be of value in relieving congestion by lowering the general blood pressure.

If the tension is relieved by medical treatment it is better to wait until the eye is quiet before operating, provided the angle of the anterior chamber has been opened. The choice of operation then

depends on the state of the angle, which should be explored gonioscopically. If peripheral synechiæ are not extensive, an iridectomy will probably suffice ; if they are, a filtration operation is advisable such as a trephining or an iridencleisis (Chapter 27).

In all cases of acute glaucoma the other eye should be operated upon by a prophylactic peripheral iridectomy if the angle is narrow, even if the eye appears otherwise normal. The occurrence of acute glaucoma in the second eye within twelve months of an attack in its fellow is so common as to render this relatively safe procedure justifiable. If both eyes are affected, both can be operated upon at the same sitting.

In absolute glaucoma if the pain cannot be relieved it is best to excise the eye. If this is refused the tension may be lowered by the operation of cyclodiathermy (Chapter 27) or the pain may be relieved for a time, varying in different cases, by a retro-ocular injection of 1.5 ml. of procaine (4 per cent.) followed seven minutes later by alcohol (80 per cent.) (p. 406) ; a firm pad and bandage are applied for twenty-four hours. If the pain recurs this treatment can be repeated. In such eyes a filtration operation is rarely effective, and the frequency with which a blind painful eye with high tension contains a malignant growth justifies its removal.

Simple Glaucoma presents an entirely different clinical picture from the acute form. While the latter occurs preferentially in women in the fifth and sixth decades, of an excitable habit and with an unstable vasomotor system, simple glaucoma occurs in either sex a decade later, affecting people who are of no specific psychological pattern but who are generally the subjects of sclerosis. While closed-angle glaucoma is characterized by premonitory symptoms and a turbulent course, simple glaucoma is quietly and slowly progressive and practically symptomless. The first type always occurs in a specific type of eye with a narrow angle of the anterior chamber. The second occurs with any type of angle; this, as is found in the population generally, is therefore usually wide. In the first type, field-defects and cupping of the disc appear late and develop rapidly ; in the second, early and insidiously. Both forms of the disease, however, lead to the same end—a stage of permanent congestion, absolute glaucoma and blindness. In both types the condition is almost always eventually bilateral.

The clinical course of simple glaucoma is characteristic. No symptoms are generally experienced although mild headaches and eye-aches may occur. An observant person may notice a defect in the visual field ; while reading and close work often present increasing difficulties due to accommodative failure owing to pressure upon the ciliary muscle and its nerve supply. An increase in the strength of presbyopic glasses is therefore often required. On the whole, however, the disease is so insidious that it is often not noticed until

the vision of one eye is almost lost and that of the other seriously impaired, when it may be discovered only by accident. This constitutes one of the main reasons why periodic eye examinations by an expert are advisable after mid-life, and certainly whenever visual symptoms require a change of spectacles. Central vision usually remains unaffected until a late stage and is no guide to the extent or progress of the disease.

Defective light sense is often an early feature of simple glaucoma. The light minimum is raised and dark adaptation is slowed, so that patients take longer to get used to the lower degree of illumination in passing into a dimly lighted room, a disability which becomes increasingly disturbing in the later stages.

The diagnosis therefore depends on the three objective signs—raised tension, cupping of the disc and field defects.

The *tension* in simple glaucoma requires careful study and repeated observation; hospitalization of the patient may be advisable over twenty-four-hour periods, at any rate in the early stages. The initial change is not so much a rise of tension as an exaggeration of the normal diurnal variation (p. 21) to form a rhythmic swing.

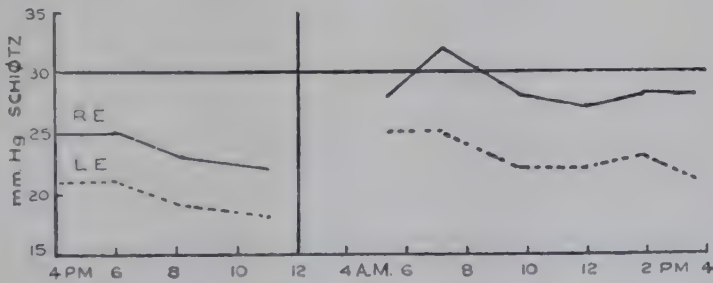


FIG. 235. The variation in tension in an early case of simple glaucoma. The abscissæ are the times of day; the vertical line indicates when the patient was asleep.

During the rising phase of tension the venous pressure in the episcleral veins may increase above the pressure in the anterior chamber as is seen by the blood influx into the aqueous veins (p. 18). Before a fall of tension, whether it occurs naturally or is induced by miotics, the opposite phenomenon is seen; the venous pressure falls and the aqueous veins fill with clear fluid. Moreover, the entire cycle is abolished by cholinergic drugs (miotics) or by blocking sympathetic activity by procaine injections into the orbit or neck to block the ciliary or stellate ganglion.

Some 20 per cent. of cases show the rise in tension in the morning, some 25 per cent. in the afternoon, and the majority shows a biphasic curve, rising at both times (Figs. 235 and 236); in most cases,

however, contrary to what happens in closed-angle glaucoma, the tension falls during the evening. At first, between its phasic rises, the tension usually returns to normal; as time goes on the variation increases and the normal level is no longer attained. The difference between the "peak pressure" and the "base pressure" thus diminishes and a permanent elevation occurs, usually, however, retaining some of the phasic element. In many cases, if this raised tension is prolonged, the eye goes quietly blind owing to atrophy of the optic nerve fibres; in other cases, if the tension is sufficiently high, a congestive phase develops; and in all cases the natural result is a condition of absolute glaucoma similar to that occurring in the acute form of the disease (*q.v.*).

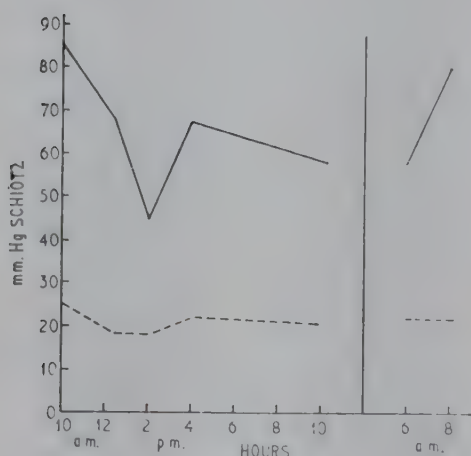


FIG. 236. Simple glaucoma in a woman aged 65. The right eye (continuous line) has absolute glaucoma with no perception of light. The left eye (dotted line) is clinically normal but the resemblance of the two curves shows that it is in a pre-glaucomatous state.

It must be stressed that in the initial stages the tension is not necessarily permanently raised; the important feature is its variability which occasionally may involve a swing as great as 30 or 40 mm. Hg (Schiötz) without causing symptoms or congestion. A single reading of the tension is therefore valueless if it is found to be normal since this may coincide with a temporary fall to a base pressure which may not be elevated (Fig. 235). *A variation in the ocular tension of over 5 mm. Hg (Schiötz) should always excite suspicion of glaucoma even although the whole excursion lies under the limits generally accepted as normal (25 mm. Hg).* This constant variation in tension can cause grave damage to the eye and produce field defects and cupping of the disc, particularly when the lamina cribrosa is weak and much vascular sclerosis is present. This clinical condition is sometimes called "low-tension glaucoma"; but indicating as it does an unhealthy and non-resistant eye, it frequently carries a graver prognosis and is more difficult to treat than an eye the function of which is maintained until the base pressure becomes high.

Cupping of the optic disc, is usually an early feature of simple glaucoma (Figs. 237-8).

The process frequently starts segmentally, often in the lower temporal quadrant corresponding with the early loss in the upper nasal field. When fully developed it differs in ophthalmoscopic appearance from a deep physiological cup, with which it is most likely to be confused, in that the excavation reaches to the edges of the disc and the sides are steep, not shelving. The retinal vessels have the appearance of being broken off at the margin of the disc. If they are accurately focused here their continuations upon the floor of the cup are slightly out of focus and look broader and paler. When the edges overhang, as is often the case, the course of the vessels as they climb the sides of the cup is hidden. By the indirect method of ophthalmoscopy slight lateral movement of the



FIG. 237. Glaucomatous cupping of the optic disc.

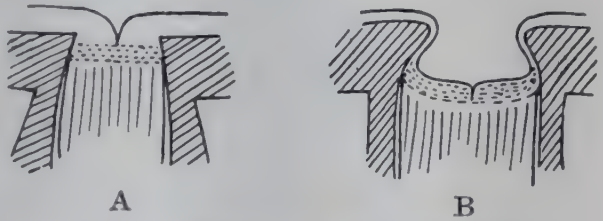


FIG. 238. A, diagram of meridional section of normal disc ; B, diagram of meridional section of glaucomatous cupped disc. Note the displacement backwards of the lamina cribrosa.

condensing lens causes a distinct parallax (p. 122), which is more marked the deeper the cup. By the direct method the difference in level between the vessels at the edge and on the floor can be measured (p. 124). When the tension is high pulsation of the arteries may be seen—always a pathological phenomenon (p. 130) (Plate X, Fig. 3).

There is always some atrophy of the optic nerve when the disc is cupped by the glaucomatous process and as the fibres degenerate the structure of the lamina may become exposed at the bottom of the cup ; it is therefore not surprising that there may be great difficulty in distinguishing a shallow glaucomatous cup from the slight depression which follows simple atrophy of the nerve without increase of tension (p. 349). In shallow glaucomatous cups the disc has a pink colour, whereas the atrophic cup is white. In many early cases all the conditions have to be weighed carefully before it is possible to come to a definite conclusion ; and in early cases progressive changes should be

carefully noted and periodically described and drawn or photographed so that any progress may be recorded rather than left to memory.

Cupping of the disc is due to two factors. To some extent the raised pressure may act mechanically, forcing the lamina cribrosa backwards and kinking the nerve fibres against the sharp scleral shelf ; the weaker the lamina the more readily this occurs. A more important factor, however, is vascular sclerosis with subsequent anæmic atrophy of the optic nerve. Such sclerosis, sometimes of marked degree, is an almost constant accompaniment of simple glaucoma. The characteristic pathological feature of the optic



FIG. 239. The optic nerve in a case of advanced simple glaucoma. Note the cavernous atrophy towards the left of the figure ($\times 25$) (Ashton).

nerve in such cases is the early disappearance of the nerve fibres without a corresponding increase in the supporting glial tissue (as occurs in primary optic atrophy) so that large caverns or lacunæ are formed (*cavernous optic atrophy*). Such a histological picture develops typically in any organ when the blood supply is gradually cut off ; the more highly differentiated elements degenerate and disappear and, owing to lack of nutriment, the supporting elements do not proliferate but eventually also degenerate (Fig. 239).

Such a condition may occur in the optic nerve without any irregular variation or rise in tension. These cases show the same appearance of

the disc and the same field defects as characterize simple glaucoma ; but they should be designated, not as glaucoma, but as *ischæmic optic neuropathy*.

The *field defects* in simple glaucoma usually run parallel to the changes in the optic disc. The careful and repeated charting of the

FIGS. 240-43. Changes in the visual fields in glaucoma.

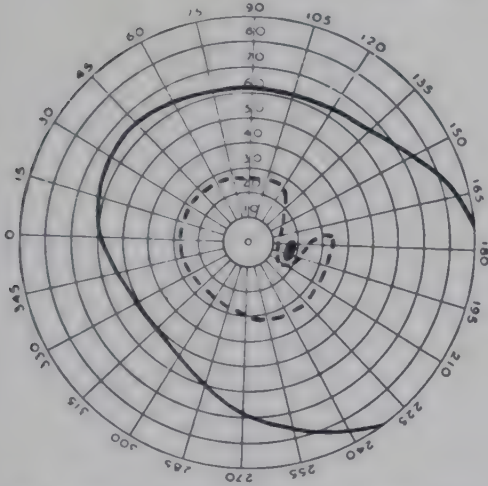


FIG. 240. Baring of the blind-spot (5/330, 1/2,000).

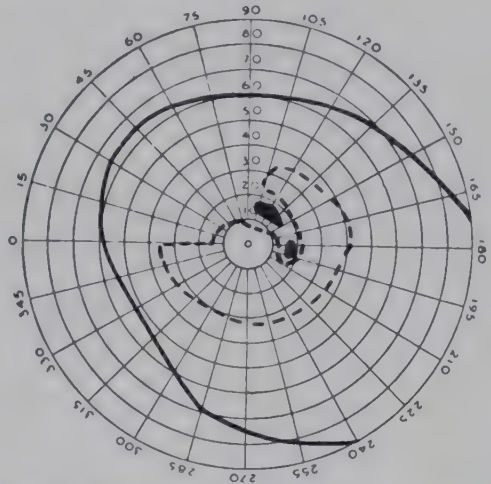


FIG. 241. Early sickle-shaped scotoma (5/330, 1/2,000).

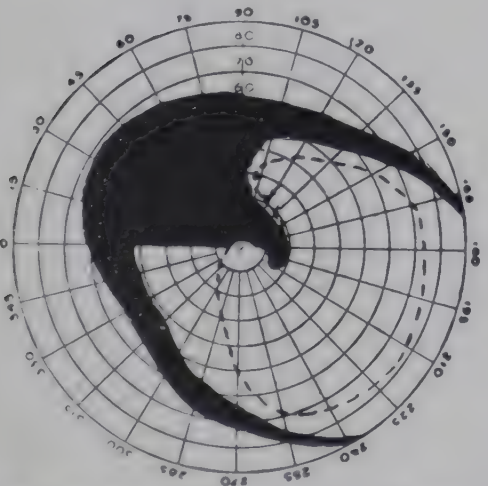


FIG. 242. Sector defect, Roenne's step and arcuate scotoma (5, 2/330).

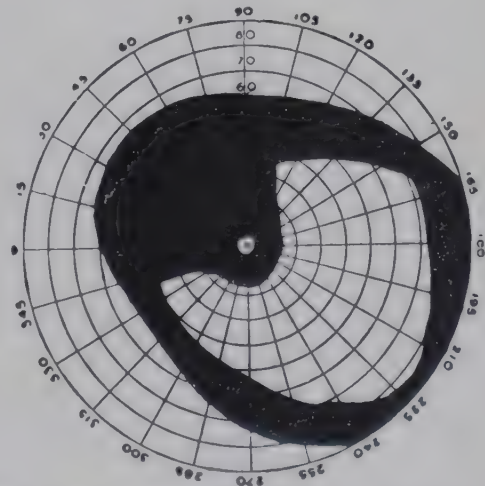


FIG. 243. Double arcuate scotoma and quadrantic defect (5/330).

fields—particularly the central fields by the Bjerrum screen (p. 136)—is of unusual importance owing to the retention of central vision until the disease is at an advanced stage (Figs. 240-3).

The earliest sign is frequently a localized constriction of the central field to very small test objects (1/2,000) so that instead of skirting the 30° isopter concentrically, the field becomes deformed,

curving inwards to exclude the blind-spot (baring of the blind-spot, Fig. 240). A second early sign is the appearance of one or more small scotomatous areas, initially a relative defect, in the same isopter as the blind-spot and usually above the disc. Occasionally there is a sickle-shaped extension of the blind-spot above or below, or both, with the concavity of the sickle directed towards the fixation point (*Seidel's sign*) ; this is of more doubtful significance. At a later stage a considerable area of relative defect can frequently be traced in direct continuity with the blind-spot (*Bjerrum's scotoma*). The scotoma may pass in an arc from the blind-spot above or below the fixation point, or may form a complete annular scotoma. Sometimes at an early stage and sometimes only late in the disease, defects appear in the peripheral field. The upper or sometimes the lower nasal field particularly tends to show sectorial defects which have a characteristically sharply defined horizontal edge (*Roenne's step*). These early defects are more easily demonstrated if the field is taken in dim illumination. In the later stages the general contraction is more marked, and eventually only a paracentral patch of the temporal field persists, central vision being abolished. The early changes are characteristic *nerve-bundle defects* and their distribution may be due to premature destruction of the thick bundles of arcuate fibres that crowd on the temporal side of the disc and run an arcuate course above and below the macula (Figs. 244-5).

Pathologically, little is evident in such eyes except the effects of generalized sclerosis and pressure. We have already noted the peculiar type of atrophy of the optic nerve. In the later stages the entire uveal tract may become extremely atrophic, only the larger vessels remaining. The angle of the anterior chamber is usually wide and peripheral synechiæ are not usually found ; but the trabeculæ are sclerosed and thickened and often heaped with pigment derived from the degenerated uvea while the efferent channels from the canal of Schlemm are sometimes sufficiently sclerosed to make the drainage of aqueous difficult.

The cause of the disease is unknown but it may be presumed in general to be due to sclerosis of the drainage channels so that they are permeable with difficulty and the base pressure rises, while a similar vasosclerosis occurs in the optic nerve and optic atrophy ensues. If such a process affects the whole globe the clinical picture produced is that of a raised tension and cupping and atrophy of the disc with field defects ; if the anterior segment of the eye is particularly affected, the factor of raised tension is the most prominent ; if the posterior segment, the condition of " low-tension glaucoma " results characterized essentially by atrophic changes in the optic nerve and defects in the visual field.

The *diagnosis* of simple glaucoma depends, as we have seen, on a close investigation of the tension—initially of its variations, eventually of its permanent elevation—and on the presence of

atrophic cupping of the disc and the typical field defects, particularly in the early stages those in the paracentral area, which should be repeatedly explored in doubtful cases in dim illumination. In the absence of an unstable or raised tension, ischæmic optic neuropathy should be diagnosed.

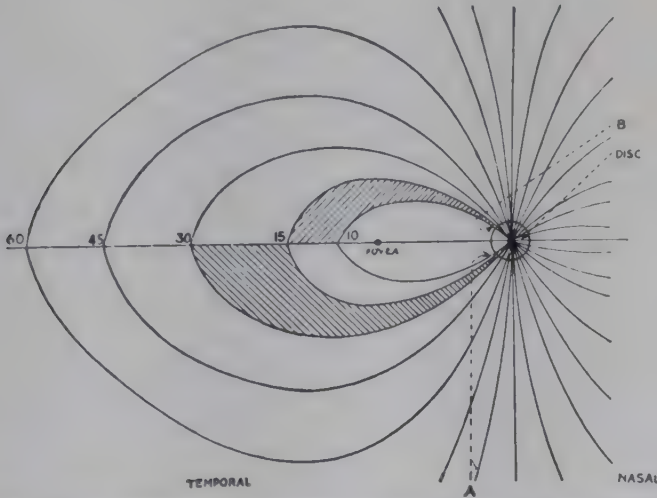


FIG. 244. The course of the nerve fibres in the retina : showing the fibres involved in field defects, A and B, in Fig. 245.

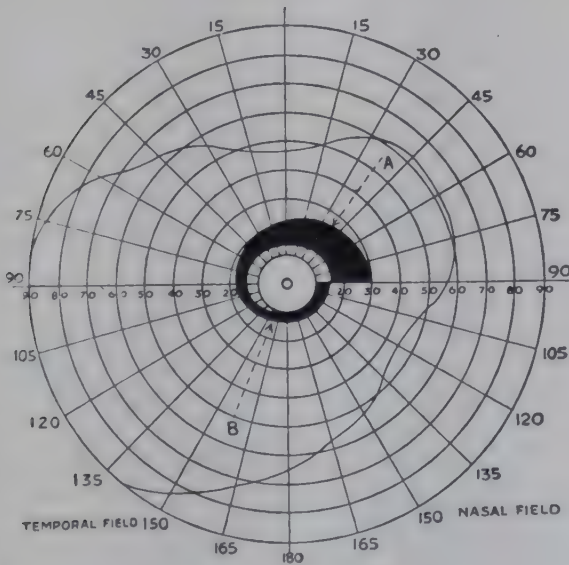


FIG. 245. Arcuate scotomata in the field of vision corresponding with lesions A and B in Fig. 244.

A most useful test of great value in assessing the progress of the disease, particularly the extent of embarrassment to drainage, is the technique of *tonography*. In this the standard Schiötz tonometer may be used or a graphic record may be obtained with the electronic tonometer. If the normal eye is compressed by a weighted tonometer

or other means for some minutes, fluid is expressed so that the ocular tension gradually falls while the compression is maintained, and when the weight is lifted the tension is considerably less than it was originally. In simple glaucoma, similar compression leads to a smaller fall during the period of compression since the aqueous cannot easily escape, and when the weight is removed the tension of the eye remains relatively high. The extent of the fall in tension gives an estimate of the facility of the outflow of aqueous.

In border-line cases a second test is the *bulbar pressure test* in which, in addition to the tonometer, a weight of 50 G. is imposed upon the globe by a spring piston (dynamometer, p. 130) placed on the sclera near the external canthus. In this way the drainage of aqueous is subjected to a greater embarrassment. A fall of less than 30 per cent. of the initial tension occurring while the eye is thus compressed for four minutes should give rise to suspicion that the drainage channels are ineffective (Fig. 246).

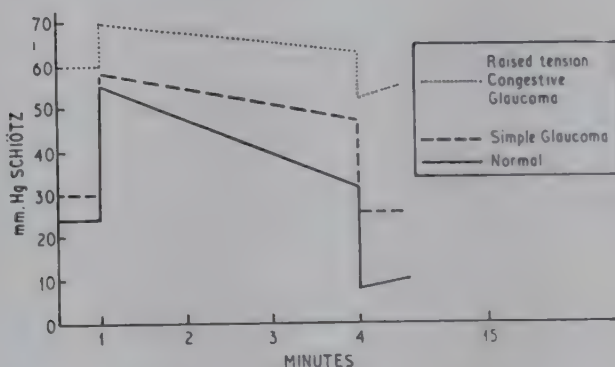


FIG. 246. The bulbar pressure test. The continuous line shows the initial rise and fall of pressure in an eye compressed with a 50 G. weight for four minutes. In simple glaucoma (broken line) the fall during the compressed period is less; in closed-angle glaucoma during a phase of raised tension (dotted line) the fall is negligible.

These tests are of great clinical value and provide an assessment of the facility of the drainage of the aqueous and therefore of the advisability of a drainage operation; they can also be used to assess the effect of such an operation already performed. It is to be remembered, however, that they all disturb the normal conditions of the ocular circulation and the results are greatly affected by variations in the ocular rigidity. It is probable that the accuracy of their results, particularly when interpreted in terms of the undisturbed eye, is not sufficient to justify some of the conclusions drawn from them as to the rate of the outflow of the aqueous; they are most safely interpreted as giving an assessment of the mechanical (organic) resistance to the outflow of the aqueous when the circulation of the eye is eliminated.

A useful provocative test is the *water-drinking test* wherein the patient drinks a litre of water before breakfast in the morning in order to lower the osmotic concentration of the blood. A rise of tension or more than 6 mm. Hg (Schiötz) should give rise to suspicion.

Treatment. As soon as simple glaucoma is diagnosed miotic treatment should be instituted. Miotics probably act largely by the mechanical action of miosis. So long as they control gross variations in tension and at the same time keep the base pressure below a reasonable level (20 mm. Hg Schiötz), and *so long as further deterioration does not occur in the visual fields*, they may be continued, provided the patient will use them regularly and submit to repeated observation. If any of these conditions are violated, an operation to control the tension by facilitating drainage should be performed. This is particularly indicated if tonography or the bulbar pressure test shows a positive result.

Pilocarpine (p. 40) is the sheet-anchor in such cases. It should be given several times a day at such times as anticipate the daily rises in tension. In early cases a 0.25 to 0.5 per cent. solution should be used as drops; the optimum strength is the weakest that will control the condition. This may be stepped up to 1, 2 or even 5 per cent., it may be combined with eserine (0.25 to 1.0 per cent.) if necessary, and if for any reason operation is inadvisable or has to be delayed, further respite may be obtained by the use of stronger cholinergic drugs such as DFP. Further lowering of the tension may be obtained by the systemic administration of carbonic anhydrase inhibitors (p. 297). These drugs are very useful as an adjunct to miotics prior to operation or to tide over a period, but owing to their side-effects their prolonged use may not be feasible.

Several operations may be employed to control the tension by the establishment of a "filtering scar." The scar in these cases is composed of spongy tissue, through the interstices of which the intra-ocular fluid is able to make its way into the subconjunctival tissue, where it is absorbed; instead of the normal drainage into the episcleral veins, the aqueous thus drains to the atmospheric level of this tissue. In an ordinary corneo-scleral section the lips of the wound are in good apposition and sound healing rapidly takes place. This is much less likely to occur if there is a gap between the lips of the wound which becomes filled with loose scar tissue so that a filtering cicatrix results. Various operations have been based upon this principle. A circular wound in the sclera minimizes this tendency and is the basis of the operation of *trephining* (p. 420). In *Lagrange's operation* (p. 422) a piece of sclera is excised; in *Scheie's operation* (p. 422) this is combined with cauterization of the lips of the wound; in *Preziosi's operation* a fistulous track is made into the angle of the anterior chamber underneath a conjunctival flap by an electrocautery. The incarceration of a piece of iris in the wound to maintain a channel is the basis of *iridencleisis* (p. 422). Other surgical alternatives are available. A communication between the anterior chamber and the suprachoroidal space, which also may lower the tension by cutting off some of the blood supply to the ciliary body, comprises the operation of *cyclo-*

dialysis (p. 422); it is suitable for low-tension cases and is of particular value in aphakic eyes. A very effective procedure is the combination in *Stallard's operation* of a flap-sclerotomy hinged on the limbus, with a small cyclodialysis and a basal iridencleisis wherein a tongue of the peripheral part of the iris is included within the lips of the scleral flap. Finally, a generalized atrophy of the ciliary body may be induced by a *cyclodiathermy* (p. 422).

Some of these operations—particularly the most effective—are not without their dangers. The presence of a draining bleb covered with thin conjunctiva may lead to the subsequent development of *secondary infection*—a serious complication, now less catastrophic if it is treated early and intensively with antibiotic drugs (Chapter 14). This is most common after a trephining operation. *Cataract* is a more common sequel, particularly if early changes are present in the lens when surgery is undertaken. If such opacities exist, an operation can be done and an extraction of the cataract performed at a later date when the corneal section is made in front of the drainage area; an iridencleisis, however, is less likely to lead to this complication, but is often less effective in lowering the tension.

In very advanced cases the field of vision may be found reduced almost to the fixation point. In these cases operation may result in the sudden complete loss of vision. Such a result is very rare but nevertheless, if the tension is not high and can be controlled by miotics, these cases are probably better left alone. If, however, the tension is not controlled, it is usually advisable to operate, but in such cases every attempt should be made to lower the tension before operation.

On the whole, it is to be remembered that more eyes are lost by delay in undertaking surgery than by surgical intervention.

All these operations undertaken for the control of simple glaucoma are uncertain in their results, for they can control the factor of tension only. In general it may be said that if the deterioration of vision is due essentially to a raised tension, its surgical relief will usually prevent further loss; if the tension is low and the deterioration is essentially due to vascular sclerosis, the vision will probably continue to fall in spite of the operation. In such cases miotics should always be continued throughout the remainder of life. General measures should also be adopted as may seem indicated to keep what remains of the ocular circulation open and slow down further sclerotic changes (nicotinic acid). Unfortunately cases of this type are not uncommon. *The prognosis of simple glaucoma is good only if the case comes under adequate control early, if operation is undertaken before raised tension has had serious effects, and if vascular sclerosis is not advanced and its progress can be delayed.* In practically all cases wherein treatment has not been instituted until a late stage, the outlook is bad. The prognosis thus depends largely on early diagnosis and the institution of early and adequate treatment.

The treatment of simple glaucoma in the congestive and absolute stages is the same as in the acute form of the disease (*q.v.*).

CHAPTER 22

DISEASES OF THE RETINA

PRIMARY affections of the retina are almost always the result of some general disease, and should therefore be properly regarded as symptomatic diseases to which the term "retinopathy" is usually more applicable than the classical term "retinitis." For this reason most of these conditions are bilateral.

These affections in general give rise to the following symptoms, only some of which need be present in individual cases. There is usually some diminution in visual acuity. There may be concentric constriction of the field of vision, or scotomata may be present corresponding with the areas especially affected. There may be metamorphopsia, micropsia, or macropsia (p. 238). The light sense is diminished, and photophobia may be present but pain is almost invariably absent, although discomfort may be experienced.

VASCULAR DISORDERS OF THE RETINA

The blood vessels of the retina are peculiarly subject to disease, sometimes in association with inflammation but more usually as a concomitant of general systemic disease.

Hyperæmia may be arterial or venous. Arterial hyperæmia, characterized by fulness and tortuosity of the arteries, accompanies not only inflammation of the retina but also inflammation of neighbouring structures, especially the uveal tract. Venous hyperæmia, characterized by dilatation and tortuosity of the veins, is the result of impeded return of blood to the heart. It may be due to general venous congestion, seen in its most extreme form in congenital malformation of the heart (*cyanosis retinæ*), or to local causes. The latter most commonly affect the veins at the optic disc, as is seen in moderate degree in glaucoma and optic neuritis, and in extreme form in thrombosis of the central vein of the retina. Increased intra-orbital pressure, as from a tumour, may also impede the exit of blood from the eye. The veins are much enlarged and dark in colour in polycythæmia and engorged in leucæmia.

Anæmia may be part of general anæmia or due to local causes. It may be sudden or slow in onset. Sudden anæmia is seen in obstruction of the central artery of the retina (*q.v.*) and in quinine amblyopia (*q.v.*) when the retinal vessels are attenuated and the optic disc is pale. The retinal vessels constrict when the oxygen concentration in the blood is high, and dilate in anoxæmia. Anæmia of slow onset is seen in atrophy of the retina from any cause and in diseases of the vessel walls. In both cases the vessels are attenuated

and, particularly in the latter, the walls become thickened and visible as white lines bordering the red blood-stream ; eventually the vessels may be transformed into white strands or may even disappear. This may be very marked in siderosis (*q.v.*).

Œdema of the Retina may be diffuse or localized. There may be general œdema, manifesting itself as a faint, diffuse haze, obscuring details so that the normal bright red appearance is replaced by a paler cloudiness, often with definite white streaks, especially along the course of the vessels, or the areas of exudation may be circumscribed. These are not pigmented and the edges are ill-defined, so that there is little danger of mistaking them for patches of choroidal atrophy. In the macular region, owing to the arrangement of the nerve fibres, œdema tends to throw the retina into radiating folds arranged like the rays of a star ; such an appearance may be very dramatic in hypertensive retinopathy and papilloœdema.

Central Serous Retinopathy presents a characteristic clinical picture of an œdema limited to the macular area ; it is caused by exudation from the parafoveal or choroidal capillaries, probably of toxic or allergic origin. It occurs preferentially in young adult males, appearing as a circular dark swelling, usually about the size of the optic disc or somewhat larger. The œdema may be pre- or sub-retinal so that the affected area is raised above the level of the retina and is surrounded by a characteristic ring-shaped reflex. The condition is usually transient and tends to resolve leaving, however, a few exudative dots and usually impairing the central vision to some extent (often to 6/9 or 6/12). Recurrences are frequent. Intravenous fluorescein injections have shown that it is sometimes due to a leakage of fluid through a defect in Bruch's membrane.

Hæmorrhages from the retinal vessels, when small, are contained within this tissue (*intra-retinal hæmorrhages*). They assume a characteristic appearance according to their location, conforming to the anatomical configuration of the layer in which they lie. When they lie superficially in the nerve-fibre layer they are striate or flame-shaped ; rounded or irregular when in the deeper layers. Intra-retinal hæmorrhages are absorbed very slowly, gradually becoming white, rarely pigmented.

When the bleeding arises from a large vessel it spreads between the surface of the retina and the vitreous (*pre-retinal or subhyaloid hæmorrhages*) ; such hæmorrhages usually occur in the neighbourhood of the macula, and may be large. At first they are round but quickly become hemispherical, the upper margin being straight owing to the effect of gravity (Fig. 247). The retinal vessels are hidden from view in the affected area. The upper part gradually becomes lighter in colour, an appearance generally attributed to the sinking of the red corpuscles, and the blood slowly becomes absorbed, usually in a patchy manner, but finally disappears, although

numerous cholesterol crystals may often be left as bright glistening spots. In this event vision is restored, but if the underlying vascular condition persists, recurrences are not uncommon. Large hæmorrhages tend to break into the vitreous rendering it opaque so that the fundus reflex is lost ; the blood may be absorbed or a proliferation of fibrous tissue may result (proliferative retinopathy, *q.v.*).



FIG. 247. Subhyaloid hæmorrhage.

Retinal hæmorrhages are due to many causes. Most frequently the vessel walls are weakened by general disease, which may be a vascular degeneration, a metabolic disturbance or a blood dyscrasia. Any of the causes leading to retinal hyperæmia may give rise secondarily to hæmorrhages. They may be due to pressure during birth in new-born infants, and are probably responsible for some so-called congenital retinal defects, particularly white and pigmented spots of atrophy at the macula and elsewhere. Similarly they occur in cases of severe compression of the thorax or neck in older people. Whooping cough may lead to retinal as well as the more common conjunctival hæmorrhages. Traumatism, including severe contusions and wounds, gunshot wounds of the eye, and of the orbit without direct injury to the eye, is another cause, and may be responsible for extensive extravasations which may burst through into the vitreous. To this category belong post-operative hæmorrhages. When the globe is opened the intra-ocular pressure is suddenly reduced to zero dilating the intra-ocular vessels and throwing strain upon their walls. If these are diseased, there is much danger of their rupturing, an accident particularly prone to affect the vessels supplying the choroid. Considering the age and condition of many patients, it is surprising that hæmorrhage is not more

frequent. One of the common causes is a tearing of the retina involving a small blood vessel by the tugging upon it of a vitreous adhesion, an event which may complicate a posterior detachment of the vitreous.

Minute hæmorrhages, unless in the macular region, may cause little obscuration of vision. The smaller spots may remain unaltered for months, although this is only apparent in some cases, old spots clearing up and being replaced by new ones. They may be absorbed without leaving any trace. Subhyaloid hæmorrhage usually abolishes central vision temporarily : it takes some weeks to clear up, the length of time varying with the size of the extravasation.

Exudative Retinopathy of Coats presents a characteristic ophthalmoscopic picture seen usually in boys, otherwise apparently healthy (Plate XII, Fig. 3). There is usually a large raised yellowish white area or several smaller areas posterior to the vessels ; detachment of the retina, cataract or glaucoma may occur in the late stages. There is always evidence microscopically of hæmorrhage between the retina and choroid and in the deep layers of the retina, and the ophthalmoscopic appearance is usually characterized by a number of small aneurysms and a varying amount of exudates, sometimes with masses of cholesterol crystals embedded in it ; the choroid is at first healthy. A very similar picture may be produced in angiomatosis. A somewhat similar disease may occur in older patients. No treatment is effective.

Proliferative Retinopathy. When a hæmorrhage occurs into the vitreous, the blood clot is usually almost completely absorbed. In some cases, however, especially in the presence of a general disease such as syphilis, nephritis, diabetes or some other form of toxæmia, when the hæmorrhage has been large it may become organized by fibrous tissue derived from the mesoblastic elements associated with the retinal vessels or on the optic disc. In this way large sheets of tissue are formed which may proliferate widely into the vitreous (Plate XII, Fig. 1). Owing to the loss of vision and the liability to retinal detachment, the prognosis is often serious.

Retrolental Fibroplasia is a disease which is generally noted some weeks after birth in premature infants who have been given high concentrations of oxygen, and is usually confined to those under four pounds in weight at birth. The earliest signs are dilatation of the retinal veins and the appearance of hazy white patches in the periphery of the retina which shortly show an indefinite proliferation into the vitreous. This is due to the formation of new vessels in the retina itself which bud into the vitreous. Their appearance is followed by the development of fibrous tissue which eventually proliferates to form a continuous mass behind the lens, appearing as a type of pseudo-glioma (p. 369). Activity may cease spontaneously at any stage so that some vision may be retained, but in many cases it progresses so that the retina is detached and the eye becomes microphthalmic. In this case, of course, vision is lost. The condition is usually bilateral.

Retrolental fibroplasia was not diagnosed as such until the late 1930's, whereafter it occurred commonly in hospital clinics dealing with premature infants, to such an extent that it became the commonest cause of bilateral blindness in children. It has been shown to follow excessive oxygenation in the nursing of premature infants in the early stages of their lives. As a result the retinal arteries and eventually the veins are obliterated, a phase followed by neovascularization. Since it has become recognized by pædiatricians that the safety limit is a concentration of oxygen of 30 to 35 per cent., the disease has practically disappeared. Prophylaxis is thus important. Once the condition has fully developed treatment is ineffective, for removal of the fibrous mass has been found to be surgically impossible.

Retinal Changes in Diseases of the Blood

Retinal hæmorrhages, often of a characteristic type, may occur in many blood diseases. It seems probable that in such diseases deficient oxygenation may lead to an increase of capillary permeability and consequent increased diapedesis.

In severe *anæmia*, whether of the secondary or primary pernicious type, the veins are frequently engorged and in the posterior half of the retina hæmorrhages appear, often with characteristic white centres, consisting of leucocytes centrally and erythrocytes peripherally (Fig. 248).

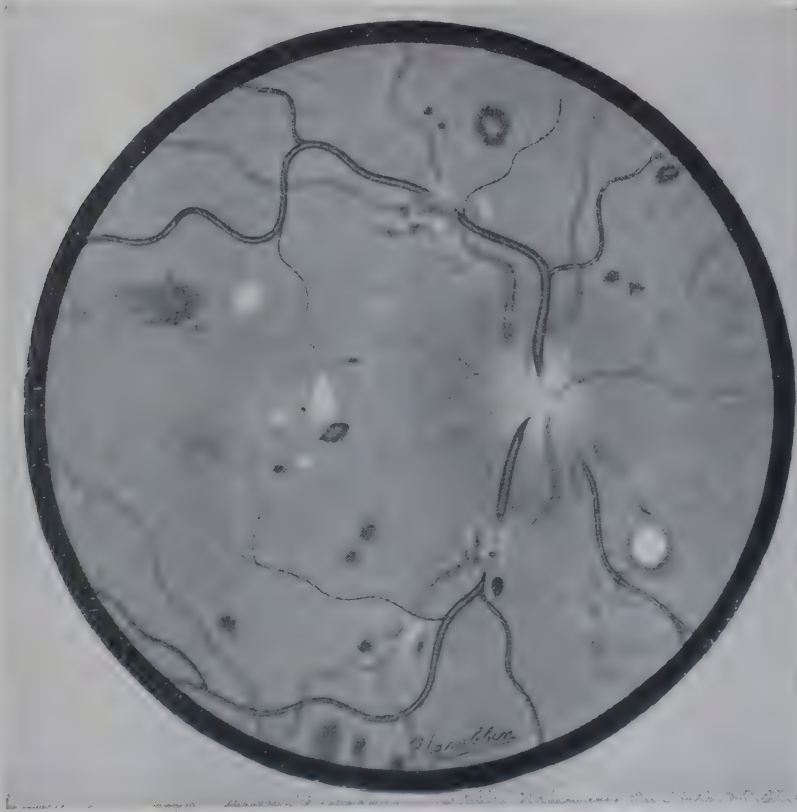


FIG. 248. Pernicious anæmia. Note the hæmorrhages with the characteristic white centres.

Leucæmia provides the most characteristic ophthalmoscopic picture among the blood diseases. The fundus is pale and orange-coloured. The veins are dilated and tortuous, often with white lines along them, and are bright red, not dark ; the arteries are small and pale yellowish red. Typical hæmorrhages occur with white centres, most commonly in the peripheral retina.

Hæmorrhages also appear in all the *purpuræ*. Similar hæmorrhages may occur in *polycythæmia vera* ; in this disease, although the arteries appear normal, the veins are enormously dilated and tortuous and the congestion may lead to the development of papilloedema.

Obstruction of the Retinal Vessels

An obstruction, complete or partial, permanent or temporary, may affect either the retinal arteries or veins.

Obstruction of the Arterial Circulation

Obstruction of a retinal artery is usually due to disease of the vessel walls ; the factor of spasm is often added which completes the occlusion. It may occur without obvious general vascular disease or when this is widespread as in arteriosclerosis, hypertension or Buerger's disease. Obstruction by an embolus, as in endocarditis, is rare, but it may occur in thrombotic carotid occlusion.

The obstruction may affect the central artery itself when the entire retina is involved or a peripheral branch when the effects are localized. *Obstruction of the central retinal artery* is nearly always at the lamina cribrosa, where the vessels normally become slightly narrowed (Fig. 249). Such an accident causes sudden and complete retinal anæmia and this tissue rapidly dies. The eye becomes suddenly blind, although when minute emboli are a factor premonitory obscurations of vision may occur. Examination of the fundus reveals a very typical picture (Plate XIII, Fig. 1). The larger arteries are reduced to threads, the smaller are invisible but the veins are little altered except on the disc where they are contracted. Within a few hours the retina loses its transparency, becoming opaque and milky-white, especially in the neighbourhood of the disc and macula. At the fovea centralis, where the retina is extremely thin, the red reflex from the choroid is visible and appears as a round "cherry-red" spot, presenting a strong contrast to the cloudy white background.

When the obstruction to the blood flow is not complete, the flow may be partially restored in the course of a few days in which case gentle pressure upon the globe may break up the column of venous blood into red beads separated by clear interspaces. The beads move in a jerky fashion through the vessels, sometimes in the normal direction of blood flow, sometimes in the opposite direction (the "cattle-truck" appearance). If the veins are easily emptied of

PLATE XII
EXUDATIVE AND PROLIFERATIVE RETINOPATHIES



FIG. 1. Proliferative retinopathy.

FIG. 2. Circinate retinopathy.



FIG. 3. Exudative retinopathy of Coats.

PLATE XIII
VASCULAR LESIONS OF THE RETINA



FIG. 1. Obstruction of the central artery of the retina.

FIG. 2. Thrombosis of the central vein.

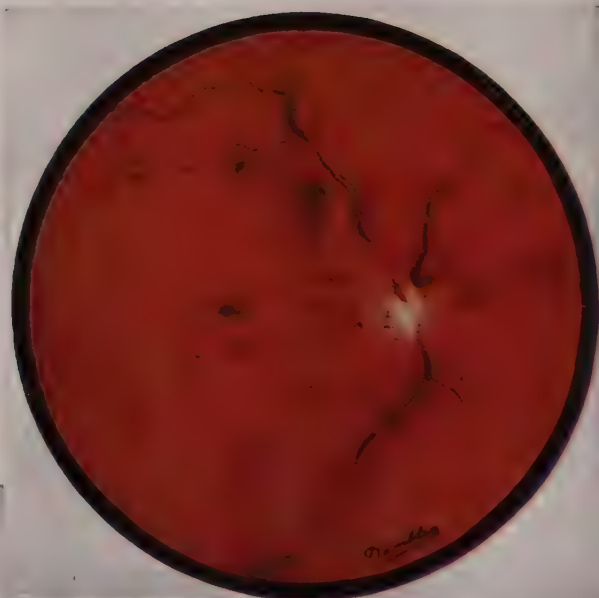


FIG. 3. Tuberculous periphlebitis.

blood or arterial pulsation is produced by slight pressure on the eyeball, it is evidence of incomplete blockage.

The white appearance of the retina takes several weeks to clear up but eventually the membrane regains its transparency and appears normal; it is, however, completely atrophic apart from the outer layers which receive their nourishment from the choroid. The vessels are contracted or reduced to white threads although some of them refill at a later stage due to the establishment of a

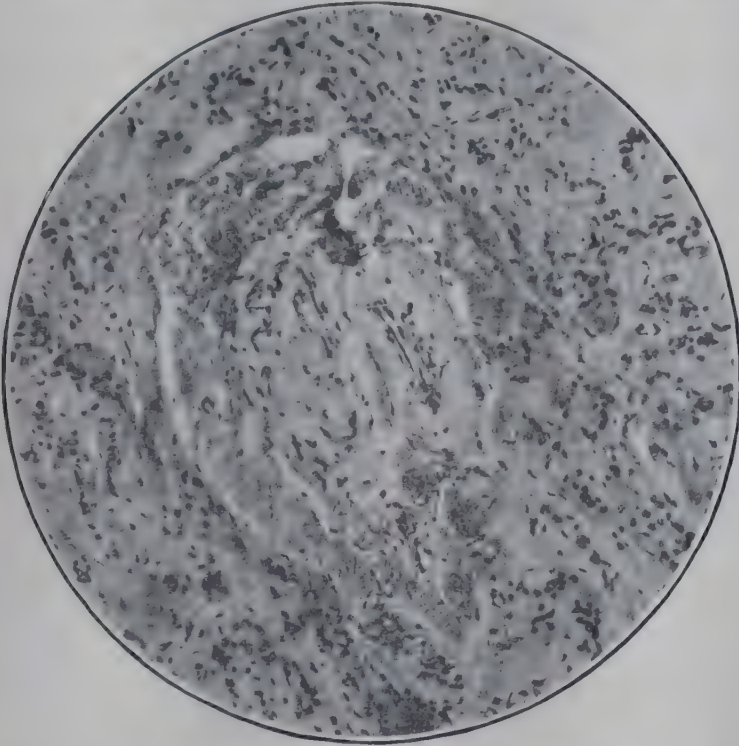


FIG. 249. Obstruction of the Central Artery (Coats). Showing central vessels posterior to the lamina cribrosa. The artery, to the left, is collapsed and obliterated by proliferation of the endothelium. The vein is concentrically narrowed by thickening of its walls, not by endothelial proliferation.

feeble collateral circulation through the anastomoses with the ciliary system round the disc. The disc is atrophic. There is no direct pupillary reaction and light perception is lost.

In some cases a certain degree of central vision persists in spite of apparent complete occlusion of the central artery. It is due to the presence of cilio-retinal arteries which, when present, always supply the macular region and naturally escape occlusion; or to a macular branch of the central artery given off proximal to the block. The remainder of the field of vision is lost. In rare cases a cilio-retinal artery alone becomes blocked.

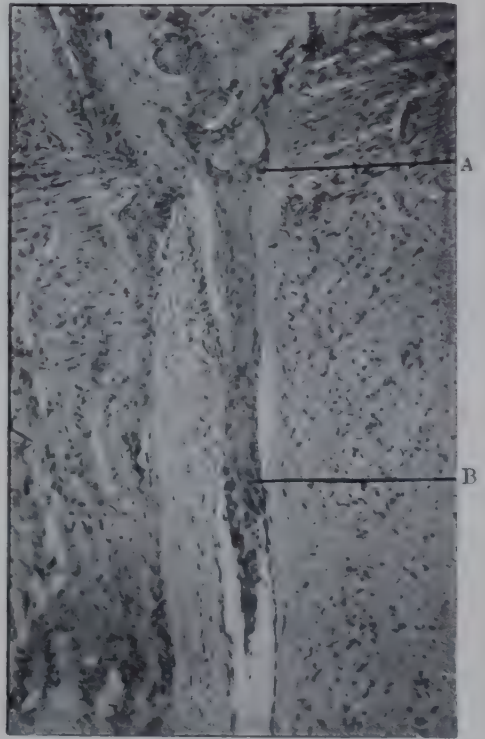
In *obstruction of a branch of the retinal artery* the area supplied by this branch is then affected alone. In the early stages the corres-

ponding scotoma is usually somewhat indefinite, but later it settles down to form a permanent sector-shaped defect.

Treatment is seldom of any avail, but attempts should be made to relieve any spasm or drive an embolus into a less important branch if the case is seen early. Massage of the globe, and paracentesis have been employed for this purpose ; to be effective such measures must be adopted without delay. Inhalation of amyl nitrite is generally useless, but a return of vision has been reported following the injection into Tenon's capsule of acetylcholine which is said to produce vasodilatation. Branch occlusion may be relieved in this way. The normal result of an occlusion of the central artery, however, is blindness.

Obstruction of the Venous Circulation. *Venous thrombosis* usually occurs in elderly people with cardio-vascular disease. In these cases the obstruction is usually in the central vein just behind

FIG. 250. Thrombosis of the central vein. Longitudinal section through the lamina (A). From this point backwards the vein is occupied by a homogeneous coagulum. At B this becomes invaded with leucocytes and ends in a sharp point lying free in the centre of the lumen, beyond which the vein is normal ($\times 200$) (Coats).



the lamina cribrosa where the vein shares a common sheath with the artery so that the two are affected by the same sclerotic process (Fig. 250). At other times in arteriosclerotic patients the block may be peripheral, usually at a bifurcation or where a sclerosed artery crosses a vein, an event which is particularly prone to occur in the superior temporal vein. In young people it may be due to an infective periphlebitis (*q.v.*) in which case a branch of the central vein is affected. Thrombosis may also be due to local causes, such as orbital cellulitis or facial erysipelas. In all cases the condition is to

be regarded as a danger signal and constitutional investigation and treatment should be assiduously undertaken.

In *central vein thrombosis* all the veins of the retina become enormously engorged with blood and extremely tortuous, and the retina is covered with hæmorrhages (Plate XIII, Fig. 2). Sight is much impaired although not so rapidly as in obstruction of the central artery, but recurrent extravasations usually destroy it. In many cases bunches of tortuous new vessels are formed upon the optic disc; in others a collateral circulation is effected by similarly tortuous new vessels in the retina. Eventually the affected retina becomes atrophic with fine pigmentary changes. The prognosis is rendered worse by the fact that secondary glaucoma ensues in two to three months in a considerable number of cases, due to sclerosis and neovascularization at the angle of the anterior chamber.

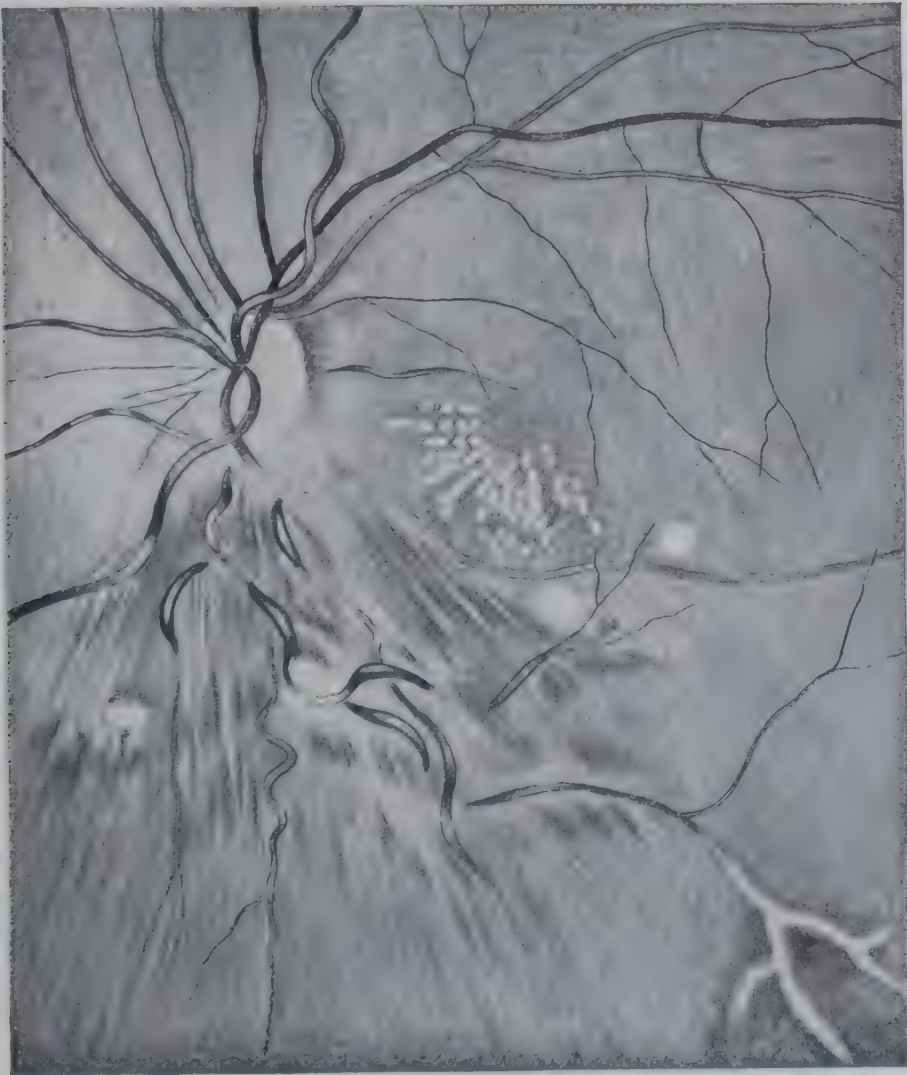


FIG. 251. Thrombosis of a branch of the central retinal vein.

In *branch thrombosis* when a single branch of the central vein is blocked, the œdema and hæmorrhages are limited to the area supplied by the vein (Figs. 251-2). In these cases the visual defect is not sectorial as in the case of occlusion of a branch of the artery ; the prognosis for central vision is better, but unfortunately blockage of the superior temporal vein frequently involves the macula. In branch thrombosis secondary glaucoma rarely occurs.

No *treatment* is effective in cases of venous occlusion once the blockage has become complete. In the early stages, however, the administration of anti-coagulants (Dindevan, etc.) may be useful in maintaining the circulation and preventing the spread of the

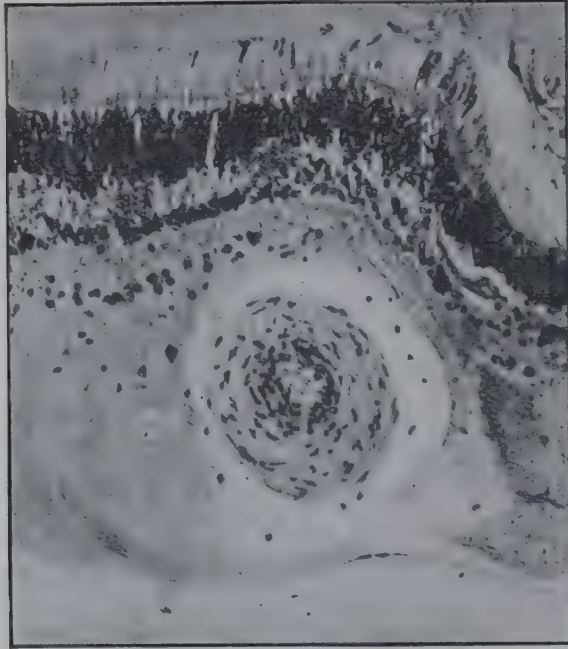


FIG. 252. Retinal vein with enormously thickened wall and narrow lumen (Coats). The perivascular space is dilated.

thrombotic process ; in arteriosclerotic patients they should be employed with care. As a prophylactic against a rise in tension, pilocarpine should be prescribed, but if a rise occurs the secondary glaucoma is difficult to treat ; if such procedures as a cyclodiathermy (*q.v.*) or injection of the ciliary ganglion with alcohol (*q.v.*) do not relieve the pain, the eye should be excised.

Vascular Sclerosis

Close observation of the retinal vessels is important since, being derived from the cerebral vessels, they closely mirror the condition of the circulation in the brain. Since they are the only vessels in the body of arteriolar calibre accessible to direct observation wherein early pathological changes can be seen, their study is of great value as a guide to the state of the circulation. Apart from

their interest as a local manifestation of disease, degenerative changes in the retinal vessels are thus of the utmost importance in general medicine. Disease of the retinal vessels is almost invariably associated with disease of the cerebral vessels, but disease of the latter may be present when there are no ophthalmoscopic signs of disease of the retinal vessels. Moreover, if marked organic changes occur in the retinal vessels, other organs, as the kidney, are probably also affected.

Before discussing the appearance of the vessels in the different types of vascular sclerosis, a summary of the most important changes may be useful.

Tortuosity of the vessels is common and may be particularly evident in the small arteries near the macula which assume a cork-screw shape. This change is of little significance unless accompanied by other abnormalities.

Alterations of the lumen may occur, appearing as irregularities in the size and breadth of the arteries so that stretches of the vessel are constricted, alternating with normal or dilated portions. These changes are due to endothelial proliferation in the intima.

Changes in the light reflex, an increase of brightness and width, are important. As we have seen (p. 127), the normal streak is due to the reflex of the blood-column seen through the transparent artery walls. When the wall becomes thickened and also reflects light, the streak becomes wider and appears as burnished copper ("copper-

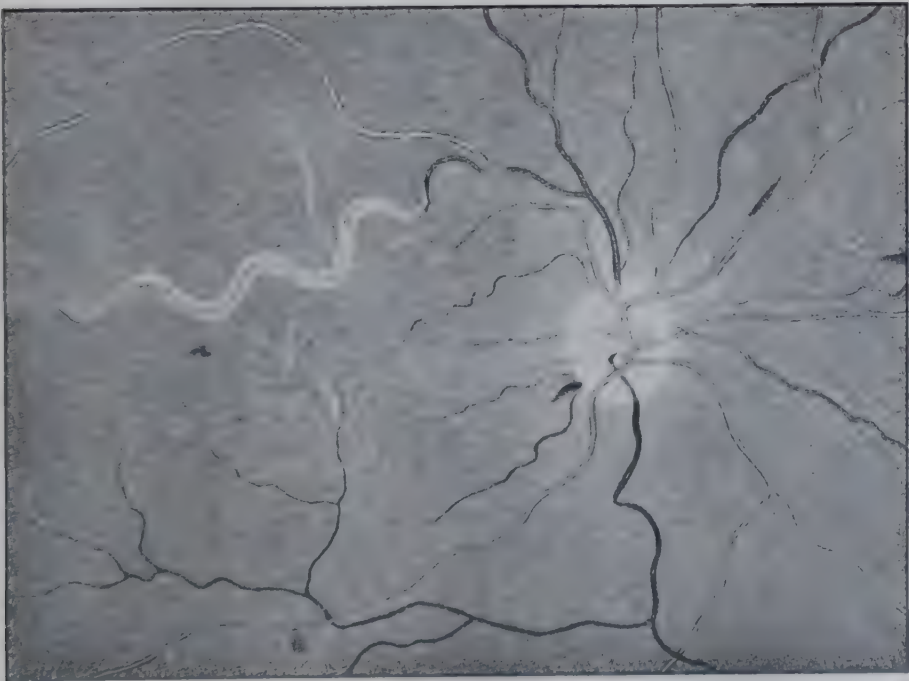


FIG. 253. "Copper-wire" and "silver-wire" arteries, degeneration of the walls of a vein, and white spots of degeneration.

wire" arteries). When the wall becomes so thick as to reflect all the light, the reflex becomes brilliantly white ("silver-wire" arteries) (Fig. 253 ; Plate XIV, Fig. 1).

Sheathing of the vessels appears when thickening of the walls makes the sides of the arteries visible as white lines.

At the *arterio-venous crossings* these phenomena are more marked. Normally it is possible to see a vein through an artery at a point of crossing ; in sclerosis the artery loses its translucency so that the vein is obscured. Moreover, the hard artery exerts pressure on the veins so that the blood flow is obstructed ; the vein seems therefore to stop at the crossing and is more distended on the distal side than on the side towards the disc (the phenomenon of *nicking*) (Figs. 254-5). Sometimes the vein appears to be pushed aside by the crossing artery ; in severe cases the vein, whether crossing above or below the artery, is diverted so that it crosses at right angles, the shortest possible route.

Minute miliary aneurysms are sometimes seen.

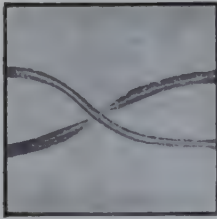


FIG. 254.

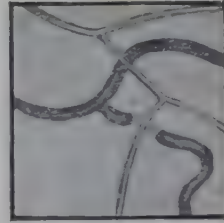


FIG. 255.

FIGS. 254-5. Compression of the veins at the arterio-venous crossing.

The various types of sclerosis seen in the retinal vessels may be classified as occurring in the arteriosclerosis of old age, and in atherosclerosis.

Involutionary (senile) arteriosclerosis (affecting primarily the muscular layer), occurs usually in patients over fifty years of age. The retinal arterioles become diffusely narrow, straight and lose their transparency, while proliferative changes may occur in the veins sometimes leading to thrombotic venous obstruction. In the absence of raised arterial tension, however, progress is slow and the prognosis relatively good.

Atherosclerosis (affecting primarily the intima) is not seen in the retinal vessels beyond the central artery and its larger branches where atheromatous plaques occasionally appear. Atherosclerotic changes in the central vessel, however, may cause an alteration in the course of the retinal vessels so that they appear to be drawn backwards into the disc ; they lose their normal tortuosity and become straight while the angulation of their branching is increased.

Plates XIV and XV
The Vascular Retinopathies

[*To face p. 320.*

PLATE XIV
THE VASCULAR RETINOPATHIES

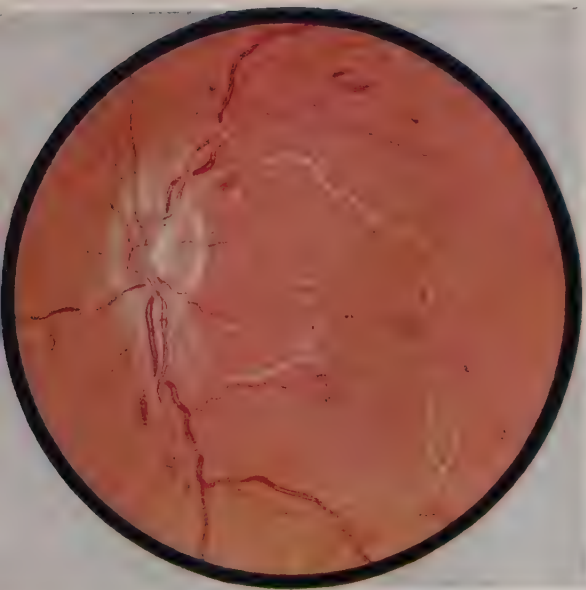


FIG. 1. Arteriosclerotic retinopathy.

FIG. 2. Diabetic retinopathy.



FIG. 3. Retinopathy in toxæmia of pregnancy.



FIG. 1. Retinopathy in arteriolar sclerosis.

FIG. 2. Hypertensive retinopathy.



FIG. 3. Neuroretinopathy of malignant hypertension.

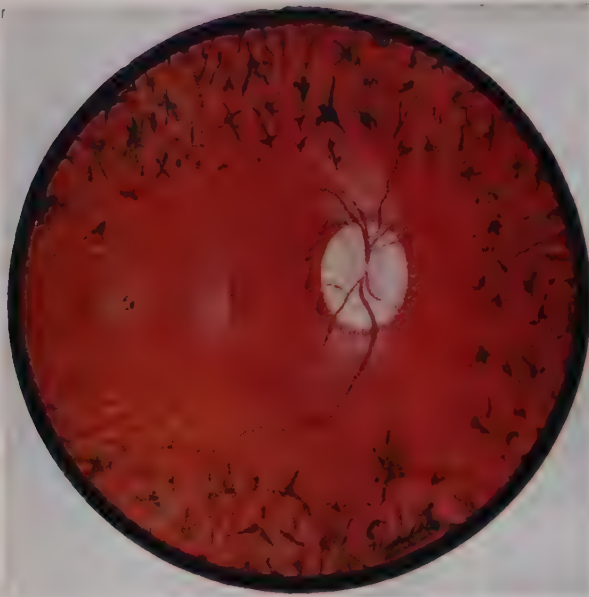


FIG. 1. Pigmentary retinal dystrophy.



FIG. 2. Retinal detachment with a hole.



FIG. 3. Congenital coloboma.

THE VASCULAR RETINOPATHIES

The vascular retinopathies occurring in general disease—hypertension, diabetes and the late toxæmia of pregnancy—are usually (with the exception of diabetes) associated with raised blood pressure and always with pronounced degenerative changes in the retinal vessels. The retinal changes probably originate from a state of anoxia which results in an increased permeability of the capillaries, the formation of multiple micro-aneurysms, and local degenerative changes in the tissues of the retina.

These circulatory changes lead to the development of retinal œdema (*q.v.*) but, since in these cases the extravasated fluid is rich in fibrin and proteins which readily coagulate, a characteristic

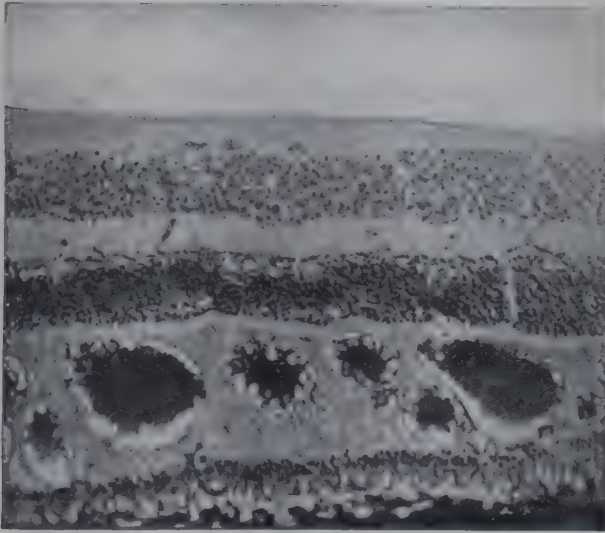


FIG. 256. Hypertensive retinopathy : star figure at macula. Note partial disappearance of outer nuclear layer.

feature is their deposition as masses of “*exudates*”. The most common form is due to œdema, swelling and degeneration of the nerve fibres (cytoid bodies), giving the appearance of cloud-like, “soft” aggregates with ill-defined margins (“cotton-wool” patches), usually seen in the superficial layers of the retina. They are commonly small, but may form accumulations larger than the disc, and, since they may disappear rapidly, they frequently change their shape. Other degenerative changes in the neural elements result in the formation of hyaline or lipid deposits which appear as discrete, “hard”, yellowish or white deposits. These are seen as round patches, and in the macular region, as with œdematous accumulations (p. 310), they tend to be thrown into radiating folds following the arrangement of the nerve fibres to form a fan- or star-shaped figure (*the macular star*) (Plate XV, Figs. 1 and 3 ; Fig. 256).

The *treatment* of all these retinopathies is confined to that of the general disease, but their presence—or the suspicion of their presence—should at once call for a medical overhaul with an examination of the blood pressure and of the urine for albumin and sugar.

Hypertensive Retinopathy may occur in four circumstances. In *simple hypertension without sclerosis*, seen in young patients, the retinal signs are few: a constriction of the arterioles which appear to be pale, unduly straight with acute-angled branching, while hæmorrhages may occur and exudates are absent (Plate XV, Fig. 2). In *hypertension with involutionary sclerosis*, occurring in older patients, the picture of *arteriosclerotic retinopathy* appears.

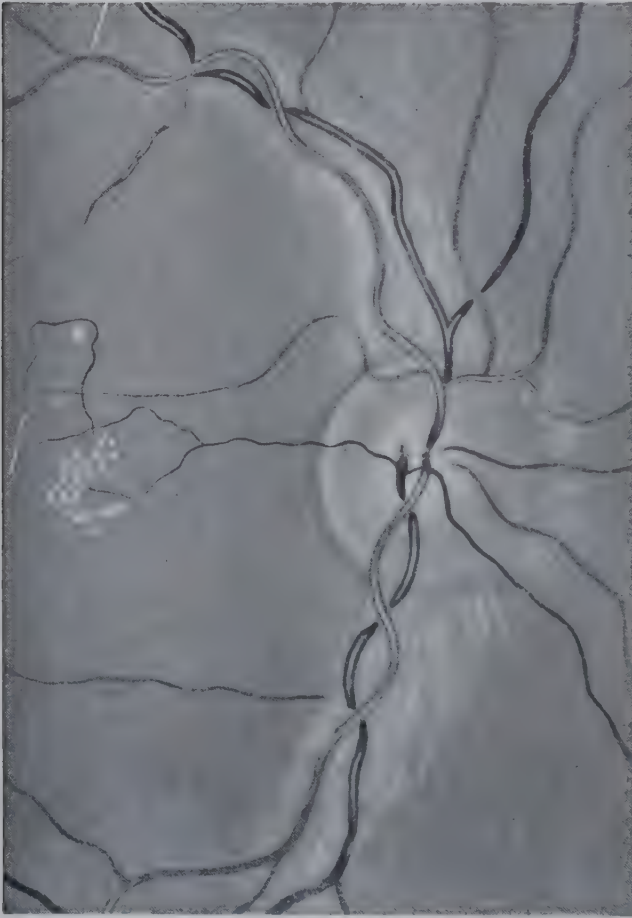


FIG. 257. Arteriosclerotic retinopathy. Compression and displacement of the veins; exudative spots at the macula (Pines).

The vascular signs just described are augmented by localized constrictions and dilatations of the vessels with sheathing of the vessels and the deposition of "hard" exudates and sometimes of hæmorrhages without any œdema (Plate XIV, Fig. 1; Fig. 257). Although the vascular changes are bilateral, the retinopathy may remain confined to one eye, and the ocular prognosis is relatively good.

In *arteriolar (diffuse hyperplastic) sclerosis* occurring in younger patients, the relatively youthful arterioles respond to the hypertension by proliferative and fibrous changes mainly affecting the

media. In the kidneys there is a chronic glomerulo-nephritis and the ophthalmoscopic picture classically known as "albuminuric" or "renal" retinopathy arises (Plate XV, Fig. 1). The vessels show evidences of hypertension. They are narrow and tortuous with nicking at the arterio-venous crossings; multiple hæmorrhages are present with, in the early stages, œdema and cotton-wool patches and, in the later, "hard" exudates scattered diffusely but usually forming a macular star. If the patient survives, these changes in the fundus may regress and although blindness does not occur the vision may be seriously impaired; the usual outcome is death from uræmia.

Malignant hypertension is an expression of the rapid progression of the hypertensive state in a patient with relatively young arteries, undefended by sclerosis. It is associated with renal insufficiency and the picture of the fundus is known as *hypertensive neuroretinopathy*, dominated by the appearance of œdema (Plate XV, Fig. 3). The entire retina may be clouded by a generalized œdema which may be particularly accentuated at the disc, resulting in a marked degree of papillœdema with multiple cotton-wool patches; "hard" exudates may be so profuse that the patches form enormous masses among which a marked macular star is usually prominent. Vision is usually gravely affected. In such cases, particularly when papillœdema is marked, the prognosis is grave and unless the hypertension can be controlled by medical or surgical methods, life is not usually prolonged more than a few months. If general treatment is successful, the ophthalmoscopic appearances may ameliorate dramatically and the vision improve; but the ultimate prognosis is unsatisfactory.

Retinopathy in the Toxæmia of Pregnancy occurs late in pregnancy, exceptionally before the sixth and practically always in the ninth month. It has many of the characteristics of hypertensive retinopathy. Initially there is narrowing of the retinal arteries, usually the nasal branches first which in any event are the smaller; and this is followed by spasmodic contractions. As the blood pressure rises, œdema makes its appearance resembling hypertensive retinopathy in its most marked forms, and the exudation may be so profuse and generalized as to cause a retinal detachment (Plate XIV, Fig. 3).

The occurrence of this disease puts a peculiar responsibility on the ophthalmologist, and any visual symptoms occurring in the later stages of pregnancy must be thoroughly investigated. Constriction, particularly of the nasal branches of the retinal artery, should at once call for extreme care in the general treatment of the case; the occurrence of arterial spasms together with an increase in weight indicating the systemic retention of fluid, are ominous signs; while the advent of retinopathy should call for a termination of the pregnancy, since its continuance will probably result in the loss of vision and perhaps of the life of the mother as well as in the birth of a still-born foetus. Timely abortion, however, as well as adequate general care, usually lead to recovery provided gross

organic changes have not occurred in the retina ; even the retinal detachment will resolve in these circumstances.

Diabetic Retinopathy is not necessarily associated with hypertension. It is frequently so accompanied, in which case the ophthalmoscopic picture may be complicated, particularly in elderly subjects, by arteriosclerosis and hypertension or even renal disease ; but in the absence of these complications a characteristic picture is seen in the fundus, particularly in young people. The causal factor is unknown ; it is not hyperglycæmia, for retinopathy occurs as frequently when the blood-sugar is relatively low as when it is high. The frequency of its incidence increases with the length of time the patient has had diabetes, even although the general disease is mild or has been well controlled ; for this reason it usually occurs in elderly patients and has become much more common since the general use of insulin which has prolonged the life of diabetics. Retinopathy is common after the disease has lasted ten years and affects the majority of patients after twenty years ; but it is not invariable. It affects young and old, for it is the diabetic age, not the chronological that is important. Nor does the presence of retinopathy bear any relation to the prognosis of the diabetes or the expectancy of life ; visual damage, however, is frequently great and permanent, a calamity since the disease is bilateral.

Ophthalmoscopically the earliest changes characteristically affect the smaller blood vessels. Small hæmorrhages are common, and the degeneration of the vessel walls leads to the development of micro-aneurysms, sometimes in vast numbers, which appear as minute round dots sometimes arranged like clusters of grapes at the ends of small vascular twigs ; these are frequently mistaken for deep hæmorrhages and may appear as an early sign in the macular area. Œdema is usually not marked, but all over the posterior pole there tend to gather hard, white or yellow, waxy-looking patches of exudates with clearly cut, often serrated margins, which occasionally coalesce into extensive plaques (Plate XIV, Fig. 2). The fully developed ocular picture with micro-aneurysms is often associated with evidences of glomerulosclerosis in the kidney (Kimmelsteil-Wilson nephropathy).

When these changes involve the macular area the central vision is profoundly affected. In addition, large hæmorrhages may break into the vitreous completely obscuring all view of the fundus ; proliferative retinopathy (p. 312) is a common sequel in which the fibrous tissue may become heavily vascularized. Rapidly developing cataract of the senile type is also frequent. From these complications many patients become blind.

No specific ocular treatment is available except that in certain early cases light-coagulation of the new vessels may tend to prevent their progression and inhibit hæmorrhages. All that can be done is the adequate and constant control of the diabetic condition. So far

as the retinopathy is concerned it has sometimes been found that the reduction of animal fats in the diet and their replacement by vegetable oils may be of some value. Destruction of the pituitary by radiation or otherwise may result in (temporary) amelioration of the ocular condition in relatively young people with no gross renal involvement when the retinopathy is of the vascular but not of the exudative type; the operation, however, is severe and the necessity for the continuous administration of systemic corticosteroids throughout life must be weighed against the possible visual advantage.

A peculiar feature sometimes met with in diabetes is *lipæmia*. It occurs especially in young patients with marked acidosis, and in the absence of adequate treatment the prognosis is grave. The ophthalmoscopic appearances are striking. The retinal vessels contain fluid which looks like milk, the arteries being pale red, the veins having a slight violet tint. The general fundus has a relatively normal coloration. Lipæmia responds rapidly to insulin treatment.

INFLAMMATION OF THE RETINA : RETINITIS

Most of the inflammations of the retina are secondary to inflammation of the choroid, the inflammatory process involving both tissues to form a chorioretinitis : these have already been discussed (*q.v.*). More rarely inflammations are primarily retinal. These may be divided into purulent inflammations caused by the pyogenic organisms and granulomatous inflammations caused by specific infections. In all cases, *treatment* is that of the general condition.

Purulent Retinitis may be either acute or subacute. The acute forms, due to the lodgment of organisms in the retina in the course of a pyæmia, lead to metastatic endophthalmitis or panophthalmitis (*q.v.*).

Subacute Infective Retinitis (*Septic Retinitis of Roth*) occurs in less virulent infections of a metastatic nature, typically in bacterial endocarditis and sometimes in puerperal septicæmia. The posterior part of the fundus is generally affected. Here numerous recurrent hæmorrhages of embolic origin appear, some of them with white centres as in the anæmias (*q.v.*). The characteristic feature is the presence of round or oval white spots (Roth's spots), many of which are cytoïd bodies. There is little general reaction in the retina although some œdema and papilloœdema may occur, but the disease is frequently fatal and vision may be seriously impaired before death.

Syphilis. Most syphilitic retinal affections are secondary to choroidal inflammations, but certain vaguely defined changes may occur primarily in the retina. *Congenital syphilitics* occasionally show a dusty discrete pigmentation of the retina at the periphery where a multitude of black and white spots appear ("pepper-and-salt" fundus). In the more definite forms there are larger atrophic and pigmented areas at the periphery (anterior retinitis), a condition often seen in interstitial keratitis (p. 211).

In *acquired syphilis* endarteritis may be prominent with whitish exudates along the course of the vessels. A diffuse retinitis may occur particularly in the secondary stage of the disease wherein the retina, especially in the central area, becomes grey and cloudy. As the condition subsides the typical picture which develops consists of an atrophic optic disc, attenuated vessels, and a generally depigmented retina with the pigment aggregated in corpuscles particularly at the periphery in a distribution resembling pigmentary retinal dystrophy (p. 327).

The subjective symptoms are defective central vision, night-blindness, irregular and concentric contraction of the field with or without central, paracentral, or ring scotomata, and metamorphopsia.

Toxoplasmosis. See p. 251.

Periphlebitis Retinæ is a relatively common disease which manifests itself clinically by repeated hæmorrhages into the vitreous. It occurs typically in apparently healthy young adults, usually males (*Eales's disease*). The ætiology is obscure, but it is probable that some cases are due to perivasculitis of tuberculous or septic origin. In many cases the source of the hæmorrhage may be found in the early stages, usually in a retinal vein near the periphery (Plate XIII, Fig. 3). The sheath is thickened, and exudates may surround the affected vessels as well as extravasations of blood. The disease has a tendency to attack both eyes in succession and to recur, so that although absorption may be complete in the early attacks, a permanent defect or complete loss of sight may ultimately follow owing to damage to the retina, dense vitreous opacities or proliferative retinopathy (*q.v.*)—a common and serious complication.

Treatment, apart from rest, should be directed to the cause. If tuberculosis is suspected or skin-tests are positive, tuberculin should be given, but the course should be prolonged for two years or more if recurrences are to be prevented; a prolonged course of corticosteroids may be advisable. If a localized area of periphlebitis is seen its destruction by diathermy or light-coagulation (p. 412) may prevent recurrences of the hæmorrhages; but it is to be remembered that fresh areas will almost certainly form unless the underlying cause can be treated, and this is frequently a difficult matter.

Retinitis from Bright Light (*photo-retinitis*) occurs after exposure of the eyes to bright sunlight, as in looking at an eclipse of the sun with inadequately protected eyes ("eclipse blindness"), or exposure to the flash of the short-circuiting of a strong current. Practically all the visible rays and many infra-red rays pass unimpeded to the retina (p. 42) and these are absorbed by the pigmentary epithelium; the pathological changes are produced by the resultant heating effect. The lesion is, in fact, a burn of the retina.

The symptoms are persistence of the after-image, progressing later into a positive scotoma, and metamorphopsia. Ophthalmoscopically

there may be no signs at first, or a pale spot is seen at the fovea with a brownish red ring round it. Later there are usually deposits of pigment and small grey punctate spots around the fovea or even the formation of a retinal hole. Prognosis must be guarded; although improvement often occurs, some defect usually remains and the scotoma may persist permanently.

No treatment is effective. Glasses impervious to infra-red rays should be used prophylactically on such occasions.

DEGENERATIONS OF THE RETINA

Senile degenerations are common in old age and are usually due to senile sclerosis of the vessels (p. 318).

Senile Macular Degeneration is usually due to obliterative sclerosis in the choriocapillaris, and is thus essentially a choroidal disease (p. 257).

Circinate Retinopathy is a more widespread degeneration dependent essentially on the same choroidal changes but involving a considerable area of the retina at and around the macula, with massive changes in the retina itself. It occurs in elderly people, particularly women. A girdle of bright white patches with crenated borders appears around the macula made up of aggregations of macrophages full of lipids (Plate XII, Fig. 2). The diameter of the girdle, which is usually an imperfect circle or ellipse or horseshoe-shaped open towards the temporal side, is generally considerably greater than a disc diameter, and follows the larger temporal branches of the superior and inferior temporal vessels. The vessels pass over the spots. The macula shows yellowish white areas, slight pigmentation and often hæmorrhages. The patches develop slowly and are usually well advanced before they are noticed. The disease is unilateral in about half the cases. Central vision is much reduced, but the field remains full. The patches sometimes disappear slowly and vision improves. No treatment is effective.

A **peripheral senile cystoid degeneration** is almost constantly present in old people, characterized by areas of atrophy associated with small cysts in the region immediately posterior to the ora serrata; it is also common in myopia. It is without significance except for the part it may play in the ætiology of detachment of the retina (*q.v.*).

Angioid Streaks. Dark brown or pigmented streaks which anastomose with each other and may be mistaken for blood vessels, are sometimes seen ophthalmoscopically. They differ in distribution from any normal set of vessels, are usually situated near the disc, at a deeper level than the retinal vessels, and are very irregular in contour. They are due to changes in the elastic tissue of Bruch's membrane, and are frequently associated with more widespread degeneration of a similar nature, as in the elastic tissue of the skin (*pseudo-xanthoma elasticum*) or of the arterial walls. Vascular and degenerative choroidal lesions elsewhere in the fundus, particularly at the macula, are common developments.

Pigmentary Retinal Dystrophy (*Retinitis Pigmentosa*) is a slow degenerative disease of the retina almost invariably occurring in both eyes, beginning in childhood and often resulting in blindness

in middle or advanced age. The degeneration affects primarily the rods and cones, particularly the former, and commences in a zone near the equator of the eye gradually spreading both anteriorly and posteriorly. The macular region is not affected until a late stage.

The symptoms are characteristic, the most prominent being defective vision in the dusk (night-blindness) (p. 359). This symptom may be present several years before pigment is visible in the retina and is due to the degeneration of the rods which are primarily responsible for vision in low illumination. The fields of vision show concentric contraction, especially marked if the illumination is reduced. In early cases a partial or complete annular or ring scotoma is found corresponding with the degenerated zone of retina (Fig. 258). As the case progresses the field becomes gradually smaller until at last it is reduced to a restricted area round the fixation point. Even although the central vision may be retained for a long time, such patients with "tube vision" have great

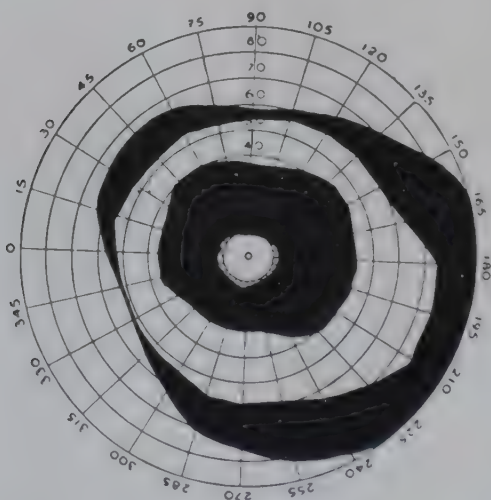


FIG. 258. Ring scotoma in a case of pigmentary dystrophy.

difficulty in getting about, for they are in much the same condition as a person looking down two long tubes seeing only the thing they are actually looking at and nothing around it. Loss of central vision does not usually occur until fifty or sixty years of age, although cataractous changes may cause earlier deterioration.

Ophthalmoscopic examination shows a characteristic picture (Plate XVI, Fig. 1). Initially the equatorial region is affected and the posterior pole and the periphery are normal, but as the disease progresses the entire retina may become involved. In the zone affected the retina is studded with small jet-black spots resembling bone corpuscles with a spidery outline. The retinal veins, never the arteries, often have a sheath of pigment for part of their course. As the pigment from the retinal pigmentary epithelium migrates into the retinal layers, the epithelium itself becomes decolorized so that the choroidal vessels become visible and the

fundus appears tessellated. The pigment spots which lie near the retinal vessels are seen to be anterior to them, so that they hide the course of the vessels. In this respect they differ from the pigment around spots of choroidal atrophy (p. 238) in which the retinal vessels can be traced over the spots. The number of pigment spots differs much in individual cases, and they are often very scanty in the early stages.

The retinal blood vessels, both arteries and veins, become extremely attenuated and thread-like. As the disease progresses and the ganglion cells become degenerate, optic atrophy sets in and gradually increases. The disc exhibits the characteristics of primary optic atrophy (p. 349), but is not quite typical of this condition for, although pale, it has a wax-like yellowish appearance. In the later stages a progressive posterior cortical cataract is formed, leading ultimately to complete opacification of the cortex.

The electroretinogram and the electro-oculogram in such cases are markedly subnormal or completely extinguished early in the disease before subjective symptoms or objective signs appear, a point of great prognostic importance in assessing the future of a young child in an affected family. In secondary retinitis pigmentosa, a sequel to an inflammatory retinitis, on the other hand, often ophthalmoscopically indistinguishable from the primary condition, the response is only slightly subnormal unless the condition is very advanced.

The condition is abiotrophic in nature and is genetically determined. In the majority of families it appears as a recessive trait and consanguinity of the parents is not infrequent. Occasionally it shows a dominant heredity when the disease may be transmitted through several generations. Exceptionally it is sex-linked. No advice can therefore be given as to the likelihood of transmission in any particular case unless the individual pedigree has been investigated.

Other defects elsewhere may be associated with the condition, the most common of which is a syndrome of obesity, hypogenitalism, mental defect and polydactyly (*Laurence-Moon-Biedl Syndrome*).

Congenital syphilis may produce a similar picture, although the distribution of the pigment spots is seldom typical (p. 325).

Treatment is eminently unsatisfactory since, despite many claims, nothing appears to have a decided influence upon the course of the disease.

Retinitis pigmentosa sine pigmento is a variety of the disease with the same symptoms, but without visible pigmentation of the retina. It is probably only the early stage of the more common dystrophy. It is progressive and leads to optic atrophy, therein differing from *congenital night-blindness*, which is a rare hereditary disease without ophthalmoscopic signs, remaining stationary throughout life.

Retinitis punctata albescens is an allied condition in which, with the same history and symptoms, the retina shows hundreds of small white dots distributed fairly uniformly over the whole fundus. A stationary form exists; but other cases are progressive and almost certainly

represent atypical varieties of the pigmentary dystrophy. In the first case the electroretinogram is normal; in the second subnormal or extinguished.

Dystrophies localized to the central area of the fundus unaccompanied by changes in the central nervous system are usually hereditary maladies. They appear at different ages. The infantile type (*Best's disease*; *vitelline dystrophy*) starts in early life with the appearance of an egg-like disc at the macula which later assumes degenerative changes. The juvenile type (*Stargardt's disease*) appears at puberty and is characterized by degenerative changes appearing as mottled pigmentary spots. The adult type (*Behr's disease*) assumes a similar appearance in mid-life, while the pre-senile and senile types are clinically indistinguishable from senile macular degeneration (*q.v.*). In all these cases central vision may be gravely impaired and no form of treatment is effective.

Familial Lipid Degenerations

Three familial syndromes characterized by lipid degeneration and the formation of large vacuolated "foam" cells may affect the retina. In two of them the ganglion cells of the central nervous system and retina only are affected (*Tay-Sachs disease* and *Batten-Mayou disease*), while the other has a more general distribution (*Niemann-Pick disease*).

Amaurotic Family Idiocy (*Tay-Sachs Disease*) occurs most commonly in Jewish children, and commences during the first year of life. Several members of a family may be affected. The apparently healthy child becomes gradually blind, with muscular weakness and wasting, and mental apathy progressing to idiocy. Death follows in from one to two years. The ophthalmoscopic picture is very characteristic and the same in every case, resembling that of embolism of the central artery but of a quite different origin. There is a round brilliantly white area at the macula, fading off peripherally into the normal fundus. In the centre of the patch is a cherry red, circular spot at the fovea. In the later stages there is optic atrophy. It is always bilateral. The disease is a primary lipid neuronc degeneration of the whole of the central nervous system including the ganglion cells of the retina, associated with profuse overgrowth of neuroglia.

Maculo-cerebral Familial Degeneration (*Batten-Mayou Disease*) may be a delayed or juvenile form of *Tay-Sachs disease*. It is a familial disease, occurring in other than Jewish children, and commencing at a later age, usually at about six or eight years. Defective vision with a central scotoma is accompanied by weak intellect, convulsions and spasticity. Ophthalmoscopically the discs are pale and the vessels small. At the macula there are yellowish-grey spots and granular pigmentation, and there may be pigmentation in other parts of the retina of the pepper-and-salt type. The ophthalmoscopic picture varies much in different cases. A cherry-red spot never develops.

Lipid Histiocytosis (*Niemann-Pick Disease*) is characterized by similar but much more widespread changes of lipid degeneration; the spleen and liver are particularly affected. Late in the course of the disease the retina may be involved when changes similar to those in *Tay-Sachs disease* may be found.

NEW FORMATIONS IN THE RETINA

The *phakomatoses* comprise a group of diseases with a familial incidence and a congenital basis with a tendency for the development of neoplasias in the central nervous system and elsewhere. In this the retina and optic nerve may be involved.

Angiomatosis of the Retina (*the von Hippel-Lindau Disease*) is a rare familial disease which generally becomes manifest in the third and fourth decades of life, more frequently in males than females. The cerebellum, medulla, spinal cord, kidneys and adrenals are also affected with angiomatosis and cysts. The



FIG. 259. Angiomatosis retinæ (Stallard).

ocular lesions are often bilateral, slowly progressive, and may precede a fatal cerebellar lesion by ten to fifteen years. The ophthalmoscopic appearances vary; the most common is a great tortuosity and dilatation of the vessels together with the presence of aneurysms (Fig. 259). Sometimes they are large like balloons; at other times small and miliary. The condition is progressive and eventually the increased permeability of the vessels leads to the deposition of enormous quantities of exudates which appear in great masses in the fundus, resembling eventually the exudative retinopathy of Coats (*q.v.*). A retinal detachment is a frequent sequel.

Treatment is unsatisfactory, but in the early stages the diathermic destruction or light-coagulation of a localized aneurysm as in the operation for retinal detachment (*q.v.*) may have a beneficial effect, or alternatively, the application of ionizing radiation (p. 370).

Tuberous Sclerosis (*Bourneville's Disease*), occurring in young individuals, is associated with nodular lesions in the central nervous system and the skin, particularly on the face (adenoma sebaceum). Similar nodular tumours about the size of the disc occur in the retina particularly near the optic-nerve head. The cerebral lesions frequently lead to epilepsy and mental deficiency.

Neurofibromatosis (*v. Recklinghausen's Disease*) may be associated with somewhat similar tumours in the retina, corresponding to those related to the nerves of the lids and orbit (p. 502).

Cysts of the Retina are commonly found in the microscopic examination of degenerated eyes, especially near the ora serrata in old people. Larger cysts occur elsewhere and are sometimes due to adhesions of folds of detached retina. In rare cases a large cyst may clinically simulate a detached retina.

Tumours of the Retina. See p. 368.

DETACHMENT OF THE RETINA (SEPARATION OF THE RETINA)

The two retinal layers—the retina proper and the pigmentary epithelium—normally lie in apposition, the potential space between them representing the original primary optic vesicle (p. 2). It is understandable that the two layers can readily become separated; such an event is called a *detachment* or (since only the inner layer of the embryonic retina is involved) a *separation* of the retina.

Detachments of the retina can conveniently be divided into two classes from the clinical point of view—*secondary detachments* due to an obvious mechanical cause when the detachment is a subsidiary event to other happenings in the eye, and *simple detachments* due to the development of a hole in the retina, in which case the state of this tissue occupies primary attention.

Secondary detachments may be due to the retina being pushed away from its bed by an accumulation of fluid or a neoplasm. The fluid may be blood (as from a choroidal hæmorrhage) or exudate; we have already considered many examples of the latter (exudative choroiditis or retinopathy, angiomatosis, the late toxæmia of pregnancy, etc.). If such an exudate is absorbed, the detached retina may well become spontaneously replaced. Tumours of the choroid (*q.v.*) have a similar effect, partly by lifting up the retina mechanically, partly by the transudation of fluid due to the circulatory disturbances caused by the mass of the neoplasm; for this reason such detachments habitually cause an extensive separation of the retina, particularly in the lower part of the eye where the fluid tends to gravitate. The diagnostic features of such a detachment are obviously important

and will be considered in the chapter on intra-ocular tumours (p. 365). Alternatively, a secondary detachment may be due to the retina being mechanically pulled away from its bed by the contraction of fibrous tissue in the vitreous, such as occurs in plastic cyclitis, proliferative retinopathy or retrolental fibroplasia (*q.v.*). The prognosis in such cases is, of course, bad.

Simple detachment of the retina is probably always due to the formation of a hole in the retina which allows fluid from the vitreous

FIGS. 260 to 262. Holes in the Retina (Shapland).

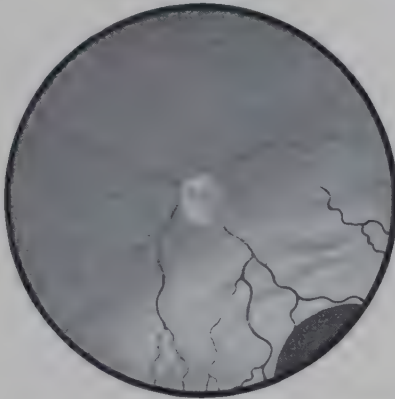


FIG. 260. Anterior dialysis.

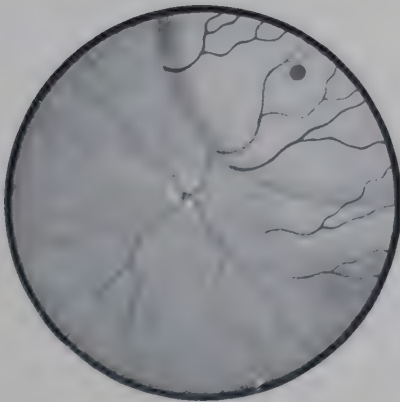


FIG. 261. Round hole.

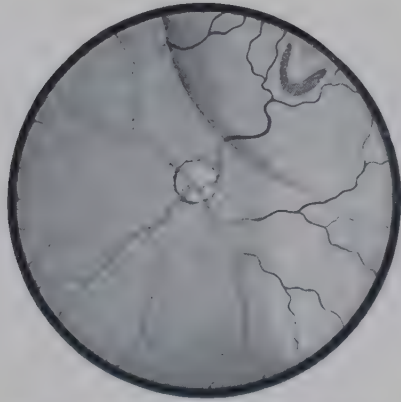


FIG. 262. Arrow-head rent.

to seep through and raise the retina from its bed. It is probable that if the vitreous gel is healthy and solid such a detachment rarely occurs; if it is fluid or detached, and particularly if it is adherent to the retina so that with movements of the eye it continually drags upon the torn area, a detachment readily develops.

Such holes or tears result from various causes. One of the commonest is the degenerative changes which sometimes occur associated with cysts near the retinal periphery, especially in myopes. It is significant that in this area the vitreous is normally

adherent to the retina and its traction on such an atrophic area or cyst readily leads either to the formation of a small hole or to a tearing of the retina at its junction with the ciliary epithelium, sometimes over a considerable area, to form a *disinsertion* or *anterior retinal dialysis* (Fig. 260). A second common precursor of a hole is a small patch of chorioretinitis, such as perhaps had given rise to no clinical symptoms. In all cases the actual mechanism of tearing is greatly aided by trauma; and it is found that trauma, often very trivial, precedes the development of a large number of detachments. In view of the ætiology it is obvious that in the young, physical strains or mechanical concussion rarely causes a detachment; this complication occurs essentially in eyes which are myopic or diseased. A detachment in a myopic or senile eye is an indication for extreme care of the other eye, which must be regarded as predisposed to the same accident. The patient should be warned against sudden and hard physical exertion, as in lifting heavy weights.

Retinal holes are frequently very difficult to find, but to find them is extremely important. In the first place, the presence of a hole designates a detachment as "simple"; in the second, cure depends on its occlusion. The shape of such holes varies; some are horse-shoe or arrow-shaped with a lid-like tongue or operculum sometimes pulled inwards by the vitreous (Fig. 262); they are most frequent at the periphery and commonest in the upper parts of the retina where the vitreous drags if it is adherent. Others are rounded (Fig. 261); usually these are less peripheral and they sometimes occur at the macula. Occasionally great and extensive tears occur; an anterior dialysis may be large in which case the choroid is seen through it and the edge of the detached retina is sharply defined.

The clinical picture of a detached retina is characteristic, but the diagnosis may be difficult in the case of shallow detachments. Failure in diagnosis is almost always due to the omission of a proper routine examination of the eye. The observer often employs the direct method of ophthalmoscopy only, with which a shallow detachment appears little altered from the normal fundus. By preliminary examination with the mirror alone, a difference in the nature of the reflex as the eye is turned in various directions will at once arrest attention, while ophthalmoscopic examination with the indirect method will usually make the matter clear.

In the early stages, and sometimes for a long period in shallow detachments, the colour of the detached portion differs little from the normal. Eventually, and sometimes rapidly, the detached portion of retina assumes a different tint from the normal fundus (Plate XVI, Fig. 2); in the most typical condition it is white or grey, with folds which show a bright sheen at the summits and appear greenish grey in the depressions. During slight movements of the eye the folds show oscillations and the retinal vessels are seen coursing over the surface, following all the curves of the folds.

Owing to the fact that they are separated from the choroid, they cut off the light reflected from this membrane and therefore look much darker than usual, and may be almost black showing no central light streak. When the detachment is very extensive great balloon-like folds may be seen, and these may cut off all view of the disc. At the edges of the detachment a considerable degree of pigmentary disturbance may appear as well as white spots of exudate, hæmorrhages, and greyish-white lines due to retinal folds. In total detachment the retina is funnel-shaped, remaining attached at the disc and at the ora serrata. Still later it becomes largely bunched behind the lens, the part attached to the disc being pulled out into a straight cord. In these cases the disturbance to nutrition of the eye leads to the development of a complicated cataract (*q.v.*) so that ophthalmoscopic examination becomes impossible.

The symptoms in the initial stages of a shallow detachment may be indefinite, for the retina may obtain sufficient nourishment from the fluid which underlies it to retain its functions only partially impaired for a considerable period. Sometimes the first symptom observed is transient flashes of light (*photopsiæ*) in a particular part of the visual field, due to slight displacements of the retina which irritate the neuro-epithelium. They should always be regarded with serious attention, but they not infrequently occur, especially in myopic eyes, without being followed by detachment.

Usually, however, the patient complains that there is a cloud in front of one eye, so that parts of objects, generally the upper or lower parts, are not seen; a scotoma is confirmed by making a chart of the field of vision. The scotoma corresponding with the detached area is usually absolute, but in shallow detachments some vision may persist. As a rule central vision is intact at first, but all detachments of the retina tend in time to be complete; when the macular region becomes affected central vision is lost and, when the detachment is total, visual disability becomes complete.

The *treatment* of a simple detachment of the retina is by operation; either by approximating the torn part of the retina to an area of the choroid in which an aseptic inflammation has been excited by diathermy, light-coagulation or cryosurgery, or approximating the choroid to the retina in this region by scleral resection, or alternatively, buckling so that in either case adherence between the two tissues results and therefore obliteration of the hole (pp. 435, 436); but before this can be done any retinal hole must be accurately located.

Since more than one hole may exist, this must involve a thorough and painstaking examination of every part of the fundus in every case; this may be time-consuming but it is necessary. Since many holes are in the extreme periphery, full mydriasis is necessary (as by the sub-conjunctival injection of mydricaine, App. I), and for this purpose the indirect method of ophthalmoscopy, using strong illumination, is

more useful and effective than the direct. Sometimes such a lesion is rendered visible only by pressing gently on the sclera near the ora serrata with a strabismus hook. A careful drawing, showing the position of retinal holes, pathological lesions, retinal vessels and other landmarks, is made of the fundus. Several examinations should be made with the patient in different postures—sitting, supine, lateral, and so on ; of these the supine is most important, since this is the position in which the operation is usually performed. Changes in posture may reveal a retinal tear which has hitherto been hidden by a retinal fold. Accurate localization of the retinal tear or holes in relation to the outside of the sclera is essential ; it is done by assessing in terms of the clock-face the meridian in which the hole lies. Its distance from the ora serrata is judged ophthalmoscopically in terms of optic disc diameters ($= 1.5$ mm.).

The prognosis in simple detachment of the retina, untreated by operation, is unfavourable. The detachment becomes total, and complicated cataract and iridocyclitis follow. The results of surgical treatment are good in 80 to 90 per cent. of cases due to trauma with a retinal dialysis at the ora serrata in the lower temporal quadrant. In healthy patients whose vitreous, retina and choroid show no disease other than changes at the site of the retinal hole the prognosis is good in about 75 per cent. of cases if operated on early. The prognosis is bad in the aged ; if the holes are large or multiple ; when the vitreous, retina and choroid are grossly degenerated ; when there is high myopia ; if the detachment has been present for nine months or more ; and always in patients who will not rest quietly after the operation.

CONGENITAL ABNORMALITIES OF THE RETINA

Congenital Pigmentation of the Retina. Small oval grey spots or groups of polygonal greyish-black spots are occasionally seen in the retina in routine examination of the fundus. They are flat, lie below the vessels, and remain unchanged indefinitely. They are congenital and due to heaps of retinal pigment any epithelium similar to those forming melanomata in the iris (*q.v.*). (See also Nævus of the Choroid, p. 366.)

Medullated Nerve Fibres. The medullary sheaths of the fibres of the optic nerve cease normally at the lamina cribrosa. Occasionally patches of fibres regain these sheaths after they have passed through the lamina cribrosa (Fig. 264). They appear ophthalmoscopically as white patches, the peripheral edges of which are radially striated, looking as if frayed out (Fig. 263). Usually the patches are continuous with the disc ; occasionally they are isolated, but rarely far from the disc. Usually the retinal vessels are covered in places by the opaque fibres. When present the blind spot is enlarged, or a scotoma corresponds with the position of the patch. Very rarely the patch is large and involves the macula, so that central vision is abolished. If glaucoma or optic atrophy causes the fibres to degenerate the medullary sheaths disappear and

no trace of the abnormality remains. It is important to be able to diagnose such fibres, since they may be mistaken for exudates, as in hypertensive retinopathy. They not infrequently occur in both eyes.



FIG. 263. Opaque nerve fibres.

They are not strictly speaking congenital, for myelination of the optic nerve progresses from the brain towards the periphery, and is not completed until shortly after birth. No treatment is required.

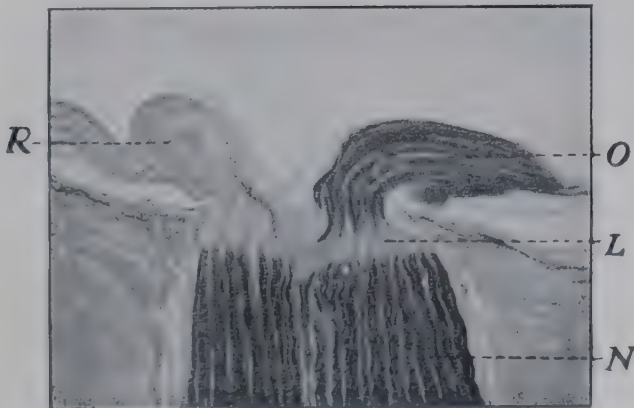


FIG. 264. Medullated nerve fibres (O), stained by the Weigert-Pal method. R, retina. L, lamina cribrosa. N, optic nerve.

Coloboma of the Retina and Choroid. See p. 260.

Albinism, see p. 261.

CHAPTER 23

DISEASES OF THE OPTIC NERVE

BEFORE discussing diseases of the optic nerve it is well to repeat that it has the structure and shares the diseases of a neurone of the second order of the central nervous system, rather than of a peripheral nerve (p. 30). The fibres (derived from the ganglion cells of the retina) have myelin sheaths normally proximal to the lamina cribrosa ; these sheaths are separated by glial tissue and a neurilemma is absent. For this reason, like tracts in the central nervous system, they do not regenerate after section.

DISTURBANCES OF THE CIRCULATION

Papillœdema (Plerocephalic œdema: Choked disc). Since the optic nerve is enclosed up to the lamina cribrosa within meningeal sheaths common to the brain, and since the subarachnoid and subdural spaces around the nerve are freely continuous with those around the brain, any rise in the intracranial pressure becomes equally evident around the nerve as within the cranium. When this occurs the subarachnoid space may sometimes become so distended that it is ampulliform just behind the globe.

As a result, œdema develops at the optic disc ; this is a purely hydrostatic, non-inflammatory phenomenon. Its cause has been in dispute. The œdema has been ascribed to several factors, but it is probably due to compression of the central retinal vein as it crosses the subdural and subarachnoid spaces, causing its collapse, whilst the thicker-walled artery continues to transmit blood. The degree of papillœdema varies with the ease of access between the meningeal spaces within the cranium and around the optic nerve, and if the optic nerve-sheath is experimentally opened in monkeys with increased intracranial pressure, papillœdema does not occur. There is often subpial œdema distal to the site of entry of the central vessels into the nerve, but not proximal to this point.

The clinical appearance of papillœdema is ushered in by a hyperæmic redness of the surface of the disc and a blurring of its margins. The blurring starts at the upper and lower margins and extends around the nasal side, while the temporal margin is usually still visible and sharp. As time goes on, progressive œdema extends over the surface of the disc filling up any physiological cup, blurring the temporal margin and spreading into the surrounding retina. As the disc swells the veins become congested and turgescient, their pulsation may be absent even on pressure upon the globe, while the small arteries also become prominent, appearing as red streaks

PLATE XVII
LESIONS OF THE OPTIC NERVE

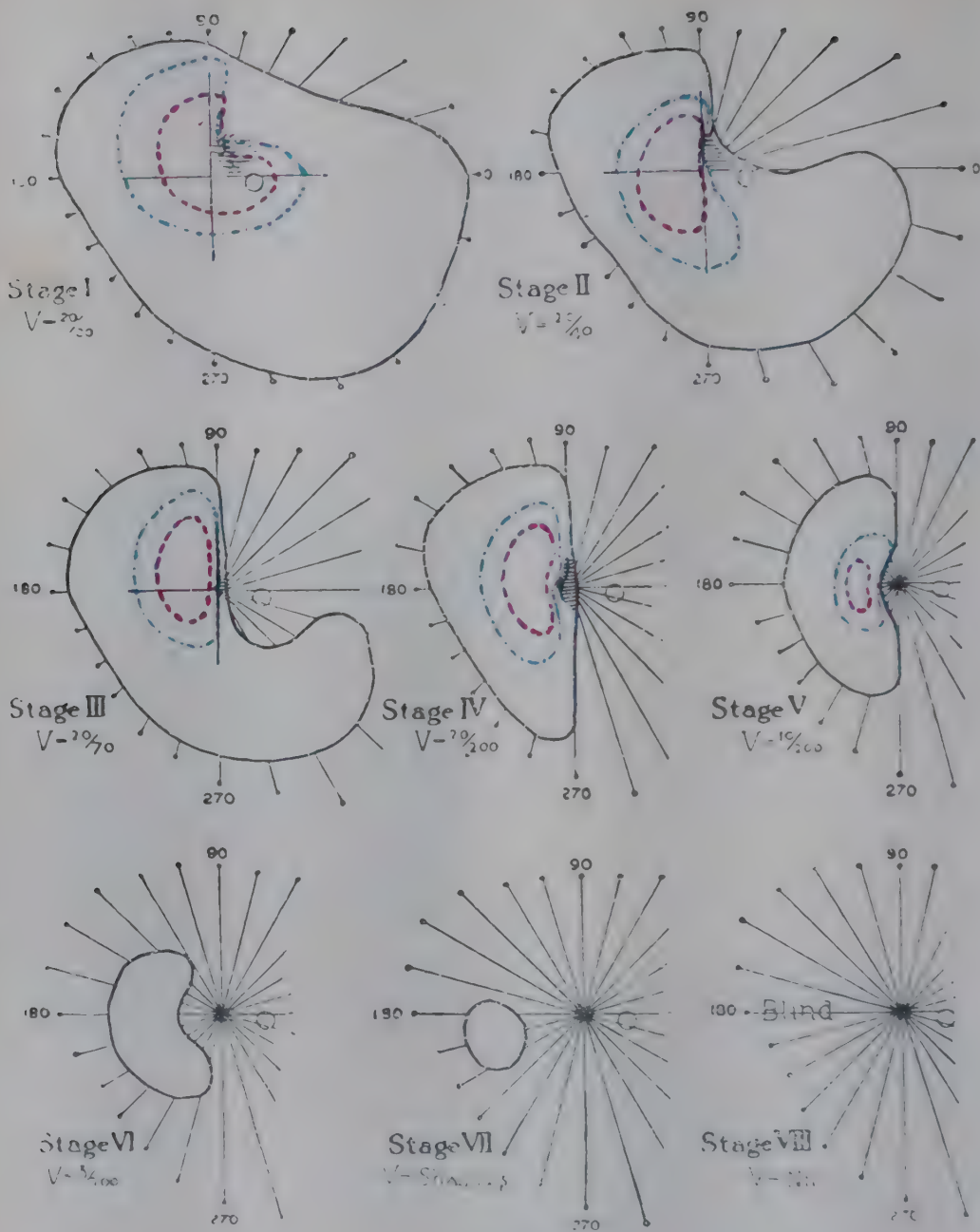


FIG. 1. Papillœdema.

FIG. 2. Primary optic atrophy.



FIG. 3. Post-neuritic atrophy.



Showing the eight stages of a progressing right temporal field defect in pituitary disease: in the first five stages, defects in the colour fields are indicated. (Harvey Cushing and Clifford B. Walker.)

on the swollen disc, sometimes giving it a striated appearance. Eventually the disc becomes elevated into a mound high over the surrounding retina and mushrooms out so that the vessels bend sharply over its margins ; with the indirect method of ophthalmoscopy a definite parallax may be elicited between its summit and the retina beneath, and by the direct method a difference of 2 to 6 or 7 dioptries may be found between the focus of the vessels at the top of the disc and those on the retina a little way off (p. 124).

Meantime, the vascular engorgement and stasis result in the appearance of numerous hæmorrhages on the disc and in the retina where they may be both flame-shaped and punctate, the œdema of the retina spreads so that this tissue is thrown into traction folds, and the veins, now tortuous and enormously dilated, may be buried for large tracts of their course in the swollen and œdematous retina (Plate XVII, Fig. 1). The surface of the disc has now lost its reddish hue and has become opaque, and exudates begin to appear upon it and in the retina itself ; the radiating, œdematous folds around the macula take on the appearance of a macular star, usually incomplete so that it takes on a fan-shape on the side towards the disc, and fluffy patches appear scattered throughout the posterior half of the fundus. At this stage the ophthalmoscopic picture may be indistinguishable from that of the retinopathy of malignant hypertension (p. 323).

Frequently the swelling begins to subside before this final stage is reached ; but in all cases subsidence eventually occurs, a process preceded by atrophic changes when the nerve fibres become unable to withstand the pressure and degenerate. When this process commences, the vascularity of the disc diminishes so that it appears a pale grey ; and eventually, even although the increase in intracranial pressure remains unrelieved, the disc becomes flat and atrophic. This appearance of "*post-neuritic*" atrophy is characteristic, depending on the fact that the absorption of the exudates is accompanied by a certain amount of organization with the formation of a variable quantity of fibrous tissue upon the disc (Plate XVII, Fig. 3). This tissue obscures the lamina cribrosa and fills in the atrophic cup. It extends over the edges, which are thus indefinite, and along the vessels as a thickening of the perivascular sheaths. Further, it throttles the vessels, so that they become markedly contracted, especially the arteries. Meantime, owing to the widespread exudative deposits, the surrounding retina often shows permanent changes, chiefly manifested by pigmentary disturbances which are most common at the macula. The amount of reactionary organization varies greatly in different cases, and the tissue laid down is in the course of time gradually absorbed to some extent. When such changes are marked they suggest the previous occurrence of papilloœdema but in their absence the conclusion that there has not been papilloœdema is not justifiable.

After any considerable papilloedema the disc rarely regains its normal appearance, but after timely relief of the intracranial pressure the changes may disappear leaving an apparently normal disc. Recurrence is rare but has been recorded.

The pathology of papilloedema shows signs of passive cedema without evidence of inflammation; in the later stages there may be some inflammatory reaction—infiltration with leucocytes, etc.—due to the irritation of the necrosed tissues. The cedema occurs first on the lamina cribrosa and peripheral parts of the nerve; the physiological cup then becomes filled in and the internal limiting membrane raised (Fig. 265). The nerve-fibres become swollen and varicose, ultimately degenerating; they show numerous cytoïd bodies, in front of but not

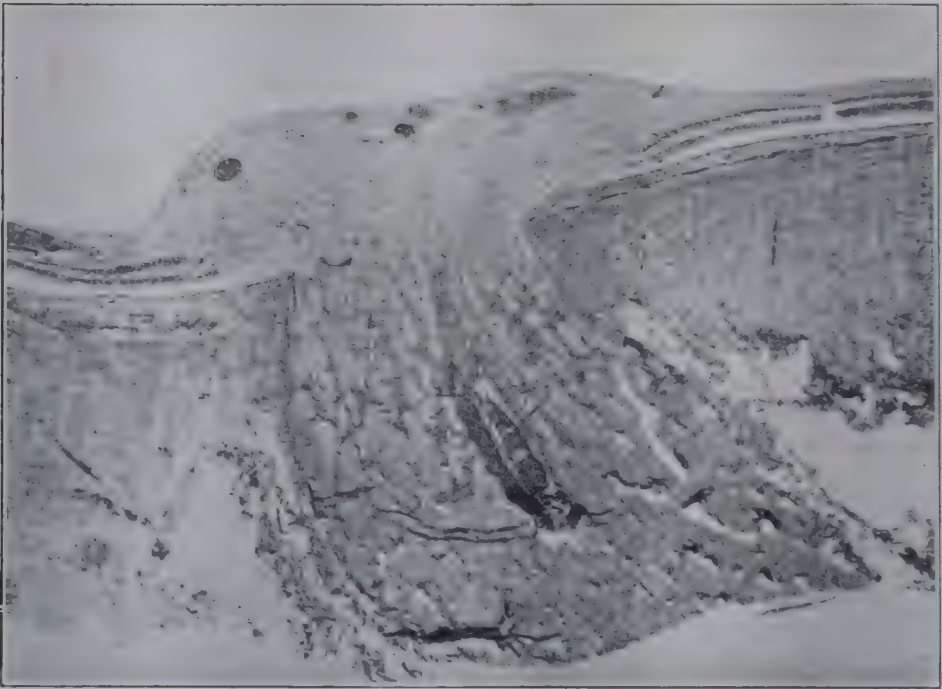


FIG. 265. Papilloedema (Coats).

behind the lamina cribrosa. The neuroglia proliferates and the mesoblastic tissue around the vessels becomes thickened; there is subpial cedema distal but not proximal to the site of entry of the central vessels, and the subarachnoid space is frequently widely distended.

The macular fan is caused by cedema in the nerve-fibre layer raising the internal limiting membrane in folds; the outer plexiform layer may be cedematous, but there are no large cystic spaces as in hypertensive retinopathy.

Symptoms are generally absent or vague and for long the vision may be unimpaired. This particularly applies to the central vision which may be unaffected even in the presence of a macular fan. Transient attacks of blurred vision, lasting for a few minutes up to an hour or so, are not uncommon in the early stages. As the

condition progresses, however, two additional signs may be found—an enlargement of the blind-spot owing to separation of the retina around the disc by the œdema, and a progressive contraction of the visual field due to atrophy of the nerves. At this stage relative scotomata first to green and red may be present. If the condition persists, however, the vision slowly diminishes but the loss bears no relationship to the amount of swelling of the disc. As atrophy sets in, complete blindness ensues ; the pupils, hitherto normal in size and reactions, are then large and immobile.

The diagnosis is easy in severe cases ; it may be very difficult in slight cases for the colour of the disc is no sure guide unless there is an undoubted difference between the two eyes. Attention should be



FIG. 266. Pseudo-neuritis in a high hypermetropie.

directed to the edges of the disc ; if these can be seen clearly defined with any lens in the ophthalmoscope there is no papillœdema, but it does not follow that there is papillœdema if they appear blurred. Astigmatism, for example, causes apparent blurring of the disc margin. Moreover, the appearances of papillœdema may be simulated in two conditions—pseudo-neuritis and a true optic neuritis. Malignant hypertension (p. 323) is itself a condition of papillœdema. In all cases attention should be directed to the amount of swelling. In the absence of other indubitable signs such as exudates or hæmorrhages, papillitis or papillœdema should not be diagnosed unless at least 2 D of swelling can be demonstrated. In some cases it is necessary to keep the patient under careful observation for a considerable period before a certain diagnosis can be made, while due attention should be paid to other signs and symptoms in the central nervous system.

Pseudo-neuritis is a condition occurring usually in hypermetropic eyes when the lamina is small and the nerve fibres are heaped up as they debouch upon the retina (Fig. 266). The ophthalmoscopic appearance of swelling and blurred margins is largely due to reflexes. The swelling is never more than 2 dioptres, there is no venous engorgement, œdema nor exudates, and the blind-spot is not enlarged.

In optic neuritis due to inflammation the appearance of the disc is often ophthalmoscopically indistinguishable from that in papillœdema. The swelling, however, is usually moderate—2 or 3 D—shelving off gradually into the surrounding retina. Vitreous opacities are usual although they may be very fine. The visual symptoms, however, are usually marked, and the acute depression of central vision and the absence of signs of an intracranial space-occupying lesion form the most important differential features.

Ætiology. In the vast majority of cases papillœdema is due to an increased intracranial pressure. Any intracranial tumour in any position, with the exception of the medulla oblongata, may induce it, the highest percentage being found with tumours of the mid-brain, parieto-occipital region, and cerebellum (p. 543). Tumours of the anterior fossa produce papillœdema more rarely and late. In general those tumours which tend to produce internal hydrocephalus are most certain to cause papillœdema. The site of the tumour is of more importance than its nature, its size or its rate of growth. Other intracranial causes—abscess, thrombosis of the cavernous sinus, aneurysm, subarachnoid hæmorrhage, hydrocephalus—are more rare; malignant hypertension is common.

In intracranial disease papillœdema is usually bilateral, although not necessarily equal on the two sides. The relative amount of swelling may be of localizing significance in these cases but its value has been over-estimated; in frontal tumours and middle ear disease, however, the swelling is usually greater on the side of the lesion. The time of onset is a more important indication than the amount of swelling, the localizing value being attached to the side first affected. Thus the swelling may be actually less on the side first affected owing to subsidence associated with commencing atrophy. Unilateral papillœdema, with or without "secondary" optic atrophy on the other side, suggests a tumour of the opposite olfactory lobe or orbital surface of the frontal lobe or of the pituitary body (the *Foster-Kennedy syndrome*, p. 542).

As rarities, conditions of stasis in the orbit may produce papillœdema—tumours of the optic nerve, a meningioma near the apex of the orbit, venous thrombosis, cellulitis of the orbit, or hæmorrhage into the optic nerve sheath.

Treatment of papillœdema is essentially the relief of the causal pressure; if this cannot be relieved, the prognosis is bad and blindness the normal result. If, however, timely decompression or removal of the tumour is successfully performed, the effect

is remarkable. Along with the relief of the general symptoms of intracranial pressure (headache, vomiting, stupor, etc.), vision improves rapidly unless the nerves have been irretrievably damaged, and the papilloedema quickly subsides; the recovery of vision, indeed, may be more rapid than the subsidence of the papilloedema. On the other hand, vision may deteriorate after operation, probably owing to progressive sclerosis at the disc, especially if surgical intervention has been delayed; if signs of subsidence and commencing atrophy are present, further diminution of vision is to be anticipated. Subsidence of the papilloedema is usually rapid after operation, a decided change being seen in a week to a fortnight, but there is considerable variation in different cases. In cerebral abscess there may be temporary increase of the swelling after operation, without, however, seriously compromising the prognosis.

In all cases the visual fields should be carefully watched and decompression should be urged from the ophthalmological point of view before peripheral constriction becomes evident. As we have seen, this indicates that the optic nerve fibres have reached the stage when they are unable further to withstand the effects of compression; once atrophy becomes clinically visible at the disc, further visual deterioration will probably follow even if surgical relief is successfully accomplished.

INFLAMMATIONS OF THE OPTIC NERVE: OPTIC NEURITIS

The optic nerve may be affected by inflammation in any part of its course, but for clinical convenience it is usual to divide inflammatory conditions into two categories—those affecting the part of the nerve ophthalmoscopically visible at the disc and therefore showing obvious signs of disease (*papillitis*), and those which attack the nerve proximal to this region and therefore show no ophthalmoscopic changes so that the diagnosis has to be made on the basis of symptoms alone (*retrobulbar neuritis*). Retrobulbar neuritis is usually divided into an acute and a chronic form; the latter is the condition which is described as toxic amblyopia (*q.v.*) and may be due to a primary retinal lesion.

Papillitis may be ophthalmoscopically indistinguishable from papilloedema (*q.v.*). The disc is at first hyperæmic, the margins become blurred, swelling and œdema ensue which spread onto the retina, the retinal veins become tortuous and much distorted, exudates accumulate upon the disc and in the retina sometimes forming a macular fan, and there are fine vitreous opacities. When the retina is seriously involved, the condition is called *neuro-retinitis*. Even in the most severe cases, however, the swelling of the disc rarely exceeds 2 to 3 dioptries.

If the inflammation has not been severe, the optic disc may appear normal on its subsidence; but if it has been sufficiently serious to

destroy the nerve fibres, the picture of *post-neuritic atrophy* results (Plate XVII, Fig. 3). Here again, the ophthalmoscopic picture is indistinguishable from that following papilloedema—the disc margins are blurred and the floor has a dirty grey colour and is filled in with organized tissue which is continued onto the constricted arteries as perivascular sheaths.

Acute Retrobulbar Neuritis, on the other hand, produces no ophthalmoscopically visible changes, unless the lesion is near the lamina cribrosa when slight evidences of papillitis may be seen with some distension of the veins and attenuation of the arteries. If atrophic changes follow, the degeneration extends not only towards the brain but also towards the eye (Fig. 267). In the milder cases pallor of the disc may be limited to the temporal side.

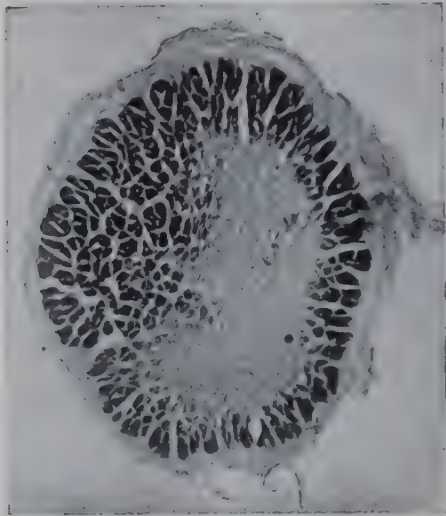


FIG. 267. Neuromyelitis optica. Transverse section of the optic nerve showing a limited area of demyelination.

The symptoms and clinical course of both types of optic neuritis are, however, very different from papilloedema, and are typical. The disease is usually unilateral and of sudden onset, when the visual loss is profound. Even in cases of papillitis it is usually much greater

than the objective signs would seem to warrant; in both types it may rapidly progress during one to eight days when blindness may be complete. In retrobulbar inflammations, this rapid deterioration of vision in the presence of a normal fundus may suggest hysteria. In these cases the diagnosis depends on the presence of local tenderness, the pupillary reactions and abnormalities in the visual fields. Recovery is often equally rapid, but the disease has a habit of showing remissions.

Local pain in retrobulbar neuritis may be felt on moving the eye. The pain is increased by pressure upon the globe, and neuralgia and headache may be present. The tenderness of the eyeball to digital pressure is limited to a small area corresponding roughly with the site of attachment of the superior rectus tendon. This sign is present only in the early stages of the disease and disappears in a few days. At first glance the pupillary reactions will be apparently normal both directly and consensually to light, as well as to convergence. More minute inspection will show, however, that although the pupil of the affected eye reacts to light, the contraction is not maintained under bright illumination so that instead of remaining contracted the pupil slowly dilates while the light is still kept on the eye. *Lack*

of sustained constriction of the pupil to light, if it can be placed beyond dispute, is of the greatest diagnostic significance.

The field of vision may show a central scotoma which may be relative for colours or absolute. It is not always quite central, but may be paracentral or sectorial or in the form of a ring around the fixation point. There is usually some peripheral loss of the field and there may be complete blindness.

Pathologically there are inflammatory changes, sometimes in the nerve (true optic neuritis), and sometimes in its sheaths (perineuritis).

The ætiology of optic neuritis is composite and it is usually impossible to deduce the cause of the disease from local signs and symptoms ; reliance must be placed on a systemic examination and even then a definite ætiological diagnosis is frequently impossible.

Much the most common cause is a demyelinating affection of the nerve as occurs in other tracts of the white matter of the central nervous system, the most common of which is multiple sclerosis (*q.v.*). The occurrence of retrobulbar neuritis should always excite suspicion of the presence of this disease, of which it frequently forms a first symptom. In these cases the habit is for recurrences to appear in either eye from time to time, occasionally at considerable intervals ; but it may be many years before more widespread signs of the disease occur.

Other diseases of the central nervous system wherein optic neuritis occurs are neuromyelitis optica (of Devic) (*q.v.*), acute disseminated encephalomyelitis, zoster, epidemic encephalitis and poliomyelitis. The more important of these will be noted in Chapter 34 but one condition wherein the optic nerve is primarily affected without other obvious central nervous involvement, deserves mention—Leber's disease.

The local causes of optic neuritis are relatively unimportant—a uveitis or a retinitis may spread onto the disc ; among the former, sympathetic ophthalmitis is prominent. Similarly, a meningitis may affect the nerve, causing primarily a perineuritis. Both syphilis and tuberculosis may act in this way. Sinus disease, particularly sphenoid and ethmoid affections and orbital cellulitis, may act similarly ; but such a sequence is probably rare.

Finally, endogenous infections may also produce an optic neuritis—acute infective diseases (influenza, malaria, measles, mumps, etc.), septic foci (teeth, tonsils), or metabolic dyscrasias (diabetes, anæmia, pregnancy, avitaminosis, starvation). The effect of exogenous toxins will be discussed under the toxic amblyopias.

Hereditary Optic Neuritis (*Hereditary Optic Atrophy, Leber's Disease*) is a form of retrobulbar neuritis, usually commencing at about the twentieth year of life. Descent is generally through an unaffected female to males, although females are also sometimes affected. Vision generally fails rapidly at first, then gradually, but remains stationary or slowly improves after six months. Both eyes are always involved.

although one may precede the other by a period varying from a few days to eighteen months. In two-thirds of the cases there is a central scotoma, either partial for colours or also for white. The peripheral field is usually normal, but concentric contraction or sector-shaped defects may occur. Total and permanent colour-blindness has been known to follow. The central scotoma generally persists, but progressive constriction of the field to complete blindness is rare. Members of the same family often show identical peculiarities in the progress of the disease. The fundus is at first normal or there is slight blurring of the edges of the disc. In later stages, after several months, optic atrophy ensues, with pallor confined to the temporal side or involving the whole disc. Apart from headache, the general health is good. The ætiology is unknown.

Treatment of optic neuritis is essentially directed to the cause, if that can be found. When the cause is obscure, mercury, iodides and salicylates have classically been prescribed with diaphoresis and heat by medical diathermy to reduce the œdema. ACTH has recently been advocated, but the results are neither consistent nor convincing. In the early stages artificial fever therapy by the intravenous injection of typhoid vaccine (p. 147) sometimes seems to hasten resolution. With all such measures, however, the effect is problematical, and the tendency to habitual remissions in the progress of the disease makes an assessment of their value difficult.

THE TOXIC AMBLYOPIAS

The toxic amblyopias include a number of conditions in which the optic nerve fibres are damaged by exogenous poisons. The most common of these are tobacco, ethyl alcohol, methyl alcohol, lead, arsenic, thallium, quinine, ergot, filix mas, carbon disulphide, stramonium and cannabis indica. In some of them (tobacco, methyl alcohol) the disease is primarily retinal and follows poisoning of the ganglion cells of the retina which results in degeneration of the nerve fibres, demonstrable only after they have obtained their medullary sheaths behind the lamina cribrosa. Others are due to a direct effect on the nerve fibres themselves.

Tobacco amblyopia results from the excessive use of tobacco, either by smoking or chewing; and also occasionally from the absorption of dust in tobacco factories. Smokers of shag and strong tobacco mixtures or cigars suffer most; cigarette smokers are rarely affected. In many cases there is also over-indulgence in alcohol but this is not invariable. The patients, usually thirty-five to fifty years of age, may have smoked excessively for years with impunity, the attack coinciding with some intercurrent cause of debility or digestive disturbance. Various substances have been regarded as the toxic agent, but a potent factor may be poisoning with the cyanide in tobacco smoke associated with a deficiency of vitamin B₁₂.

The patient complains of increasing foggy vision, usually least marked in the evening and in a dull light. Central vision is

greatly diminished, so that reading and near work become difficult ; although the condition is bilateral, one eye is usually more affected.

The fundus is normal or a slight temporal pallor may be seen in the disc ; but the diagnosis is made from the characteristic defects in the central fields. These involve primarily the centrocæcal area between the fixation point and the blind-spot. Here, occupying a horizontally oval area, there is a relative scotoma to white and colours, particularly for red, and in it, on the horizontal meridian, there are one or more islands of complete visual loss. The scotoma gradually extends to involve the fixation area itself so that central vision may be lost but the peripheral field remains unaffected.

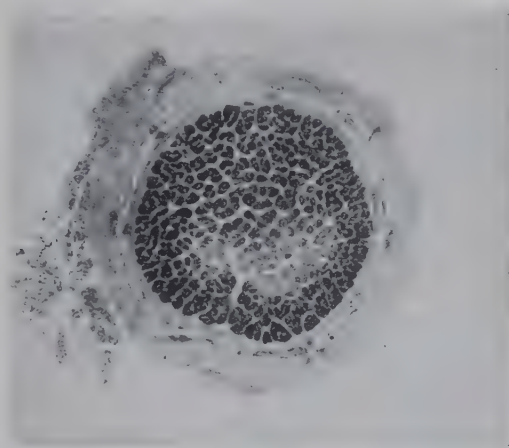


FIG. 268. Optic atrophy in tobacco amblyopia (Behr).

Pathologically the condition is due to degeneration of the ganglion cells of the retina, particularly of the macular area where the cells show vacuolation and Nissl degeneration. In the nerve the papillo-macular bundle is degenerated (Fig. 268).

Treatment consists of abstention from or severely curtailing the use of tobacco and alcohol. If this is done the prognosis is eventually good although visual improvement may not be evident for a period of some months ; thereafter it may be slow. Improvement may be hastened by large doses of vitamin B preparations (thiamine and particularly B_{12}). Recurrences are very rarely seen even if tobacco is resumed in strict moderation, but this is perhaps unwise.

Ethyl Alcohol. Although alcohol is usually an adjuvant in tobacco amblyopia, it may cause a similar amblyopia in the absence of the latter. Such patients frequently suffer from alcoholic peripheral neuritis. The disease, characterized by a central scotoma, may be due essentially to avitaminosis owing to chronic lack of nourishment (*q.v.*).

The amblyopia produced by *diabetes*, *carbon disulphide* (seen in the rayon industry), and *iodoform* resembles that of tobacco.

The amblyopias produced by *methyl alcohol*, *arsenic*, *lead*, *nitro-* and

dinitro-benzol, differ from the above type in the more serious optic atrophy which generally ensues. There is probably always a stage at which a central scotoma is present, but it is often missed.

Methyl alcohol poisoning from drinking wood-alcohol has always been common in countries during prohibition, and occurs sporadically from drinking methylated spirit. Individual susceptibility is marked. It may occur in an acute or chronic form. In the acute form nausea, headache and giddiness are followed by coma. If the patient survives, vision very rapidly fails, passing through the stages of contracted fields and absolute central scotomata to blindness. The vision may improve, but usually again relapses, becoming gradually abolished by progressive optic atrophy. Restoration is rarely complete. Ophthalmoscopically there may be blurring of the edges of the discs and diminished size of the vessels in the early stages. Later there are signs of optic atrophy, usually of the primary type (p. 349). Pathologically there is widespread degeneration of the ganglion cells of the retina.

Arsenic is specially liable to cause optic atrophy, usually total, when administered in the form of pentavalent compounds such as atoxyl or soamin. These were used for attacking the trypanosome of sleeping sickness, but have now been abandoned. In the salvarsan group the arsenic is in trivalent combination and is less toxic.

Lead poisoning is rarely seen since precautions have been taken to eliminate salts of this metal from pottery glazes, etc. The ocular signs are optic neuritis or optic atrophy, which may be primary or post-neuritic. Some cases develop a retinopathy which may be due directly to lead or be of the renal type, secondary to lead nephritis.

Quinine amblyopia differs in some striking characteristics from tobacco amblyopia. Here total blindness (amaurosis) may follow the use of the drug, even in such small doses as 1 g in susceptible persons; 2.5 g is the maximum amount of sulphate of quinine which should be given within twenty-four hours. The largest doses are usually taken for malaria, but quinine is also used as an abortifacient. The pupils are dilated and immobile. Deafness and tinnitus are present. Ophthalmoscopically the retinal vessels are extremely contracted and the disc is very pale; oedema of the retina has been described in the early stage. Occasionally blindness is permanent and optic atrophy ensues. In less marked cases or at a later stage the fields of vision are much contracted but central vision may be completely restored so that tube-vision results. The discs may remain pale for years or become normal.

A similar amblyopia may follow the use of some *barbiturate compounds ethyl-hydrocuprein* (optochin) (formerly given for pneumonia), and occasionally the *salicylates*, when the symptoms are not so severe. *Filix mas* (used as an anti-helminthic) may cause amblyopia in excessive doses, especially if given with castor oil; optic atrophy may supervene.

Treatment of these amblyopias is cessation of the drug and administration of vasodilators (amyl nitrite, sodium nitrite, retrobulbar injections of acetylcholine, etc.).

Deficiency Amblyopia. A deficiency of vitamins in the diet, particularly of thiamine, may be responsible for the development of an optic

neuritis usually of the axial type, resulting in the loss of central vision ; similar lesions in the mid-brain cause various types of ophthalmoplegia (acute hæmorrhagic anterior polioencephalitis of Wernicke, p. 542). Such an amblyopia is seen in extreme degrees of pellagra and, in association with other grave disorders of vision, was relatively common among the starved prisoners of war incarcerated by the Japanese in the Malayan and Pacific campaigns in 1943-5. An optic atrophy, usually partial but occasionally apparently complete, may eventually develop and in severe cases the prognosis is bad. An efficient diet, if resumed before atrophy develops, is curative ; after atrophy has set in the visual defect is permanent.

DEGENERATIONS OF THE OPTIC NERVE : OPTIC ATROPHY

Optic Atrophy is the term usually applied to the condition of the disc when the optic nerve is degenerated. It has been pointed out that injury to the nerve fibres in any part of their course from the retina to the lateral geniculate body leads to degeneration not only on the proximal (cerebral) side—as might be anticipated for afferent fibres—but also on the distal (ocular) side (p. 344). Optic atrophy therefore follows extensive disease of the retina from destruction of the ganglion cells, as in pigmentary retinal dystrophy or occlusion of the central artery ; these cases are sometimes called *consecutive atrophy*. The break in continuity of the fibres may be at the disc itself, such as results from the strangulation occurring in papillitis, neuro-retinitis or papilloedema. These cases are distinguished as "*post-neuritic*" *atrophy*. It also follows destruction of the nerve in the orbit, as in fracture of the base of the skull or severe retrobulbar neuritis. In addition there is a group of cases in which optic atrophy occurs without local disturbances but associated with general disease, usually of the central nervous system, or without discoverable cause. Such cases are described as *primary atrophy*. *Toxic atrophy* has already been discussed, as well as *glaucomatous atrophy*.

The essential ophthalmoscopic features of optic atrophy in general are an alteration in the colour of the disc and changes in the blood vessels. The disc is always pale, but may show varieties of tint especially associated with various types of atrophy. The pallor affects the whole disc and must be carefully distinguished from the white centre, often encroaching upon the temporal side, due to physiological cupping. The pallor is not due to atrophy of the nerve fibres, but to loss of vascularity owing to obliteration of the vessels ; it is thus an uncertain guide to visual capacity.

When the atrophy is due to disease or poisoning of the second visual neurone proximal to the disc so that there are no ophthalmoscopic evidences of previous local inflammation, it is called *simple* or *primary atrophy*. In such cases the disc is grey or white, some-

times with a greenish or bluish tint (Plate XVII, Fig. 2). The stippling of the lamina cribrosa is seen; the edges are sharply defined and the surrounding retina looks normal. Owing to the degeneration of the nerve-fibres there is slight cupping (atrophic cupping) which must be carefully distinguished from glaucomatous cupping; it is shallow and saucer-shaped, as shown by the slight bending of the vessels, but is scarcely measurable with the ophthalmoscope. There is no retraction of the lamina cribrosa. The vessels are normal or only slightly contracted.

The classical cause of primary atrophy is tabes (*q.v.*) in which the degeneration is due to a chronic inflammation of the pia which causes a secondary degeneration of the nerve fibres commencing in the optic nerve in the neighbourhood of the chiasma. Tabetic optic atrophy is slowly progressive and the prognosis is bad, but owing to efficient anti-syphilitic treatment, the disease has now become relatively rare. The same applies to the atrophy of general paralysis. The most common cause to-day is multiple sclerosis wherein recurrent attacks of transient inflammation cause an increasing degree of atrophy, but in this disease it is rarely complete. Other causes are the demyelinating diseases already noted in the ætiology of optic neuritis (p. 345), Leber's disease, and the many exogenous poisons which give rise to toxic amblyopia.

Secondary atrophy which has exactly the same ophthalmoscopic picture as the primary variety, follows any injury or direct pressure affecting the visual nerve-fibres in any part of their course from the lamina cribrosa to the geniculate body. It is noteworthy that a brain tumour will produce a secondary atrophy if it presses upon the chiasma or optic nerve, and a post-neuritic atrophy if it causes papilloedema by increased intracranial pressure. The differentiation does not indicate the nature or site of the pressure; it merely differentiates whether the atrophy has affected a normal disc or one which has been choked.

The characteristic ophthalmoscopic picture of *post-neuritic atrophy* has already been described (p. 339) (Plate XVII, Fig. 3).

In the "*consecutive*" atrophy of retinal and choroidal disease, the disc has a yellowish waxy appearance, the edges are less sharply defined, and the vessels are very markedly contracted, sometimes almost to the point of disappearance.

In total optic atrophy the pupils are dilated and immobile to light and the patient is blind; when unilateral the consensual reaction to light is exaggerated. In partial optic atrophy central vision is depressed and there is concentric contraction of the field, with or without scotomata, relative or absolute, according to the cause. It is important to note that no deduction as to the amount of vision can be made from the ophthalmoscopic appearances for the presence of all the signs of atrophy is not inconsistent with a certain, sometimes a considerable, amount of vision.

No *treatment* is effective for optic atrophy ; the prognosis depends on the possibility of controlling the causal factor.

Tumours of the Optic Nerve. See p. 526.

CONGENITAL ABNORMALITIES OF THE OPTIC DISC

Coloboma of the Optic Disc. This occurs in two forms, one of which is common, the other rare. The common form is due to incomplete closure of the embryonic fissure, and manifests itself as an *inferior crescent*, much resembling the myopic crescent (*q.v.*), but situated at the lower edge of the disc. It occurs most commonly in hypermetropic and astigmatic eyes, which are often found to have slightly defective vision in spite of the correction of any error of refraction. It is often slightly ectatic (*conus*).

In what is commonly known as *coloboma of the disc* (or nerve sheath) there is greater failure of the embryonic fissure to close. The disc looks large and the vessels have an abnormal distribution, appearing only above or irregularly round the edges. The apparent disc is really the sclera and the inner surface of the sheath of the nerve, the nerve itself being usually spread out as a pink horizontal linear band at the upper part. The floor of the coloboma is white and measurably depressed, often quite ectatic. The eye usually has defective vision.

Rarer anomalies allied to coloboma are round cavities ("holes") on the disc, generally situated in the temporal portion ; they usually look grey or black owing to shadow and patches of *pigment* due to the inclusion of retinal pigmentary epithelium.

Mention has already been made of an excess of *fibrous-like tissue* on the disc and extending a short distance along the vessels, the remnants of the sheaths of the hyaloid vessels (p. 284). Sometimes the fibrous tissue takes the form of a delicate semi-transparent membrane covering the disc and appearing to be slung from the vessels.

CHAPTER 24

SYMPTOMATIC DISTURBANCES OF VISION

APART from the disturbances of vision which have already been considered and have their origin in the eye itself, there are others dependent upon lesions in the visual nervous paths. Since they not infrequently closely simulate the disorders due to peripheral causes or are early evidence of disease, they lead the patient to consult an ophthalmic surgeon. There are also visual defects the cause and seat of which are imperfectly elucidated ; although some are probably peripheral in origin, it will be convenient to consider them here.

Hemianopia. Hemianopia denotes loss of half of the field of vision. The commonest clinical form is *homonymous hemianopia*, in which the right or left half of the binocular field of vision is lost, owing to loss of the temporal half of one field and the nasal half of the other, a condition due to a lesion situated in any part of the visual paths from the chiasma to the occipital lobe. A focus of disease in this area causes loss of vision of the corresponding halves of each retina (hence the designation homonymous) and therefore loss of the opposite halves of the visual fields (Fig. 269). Right hemianopia is more quickly discovered than left owing to the fact that reading is impossible ; left hemianopia is often discovered when the patient does not see food on the left side of the plate.

In many cases of hemianopia the fixation area in each field escapes, especially if the lesion is near the occipital cortex ; in infrageniculate lesions the fixation point is usually bisected. In a number of cases it is probable that the macular fibres are in fact spared owing to their widespread but segregated course in the optic radiations and their separate representation in the occipital pole. The immunity of the macula in vascular lesions of the cortex is attributed to the fact that the occipital pole is supplied by the posterior and middle cerebral arteries both of which are seldom blocked at the same time. The explanation in other cases is not so obvious. In certain cases the sparing of the macula may be only apparent owing to a functional shift of fixation towards the seeing part of the retina, while in other cases a possible explanation may be sought in the integrative powers of the central visual mechanism.

Lesions of the lateral geniculate body cause homonymous hemianopia (Fig. 269) ; those limited to the pulvinar and superior colliculus do not.

Cortical and Sub-cortical Lesions. The majority of cases of hemianopia is due to lesions above the primary visual centres, usually

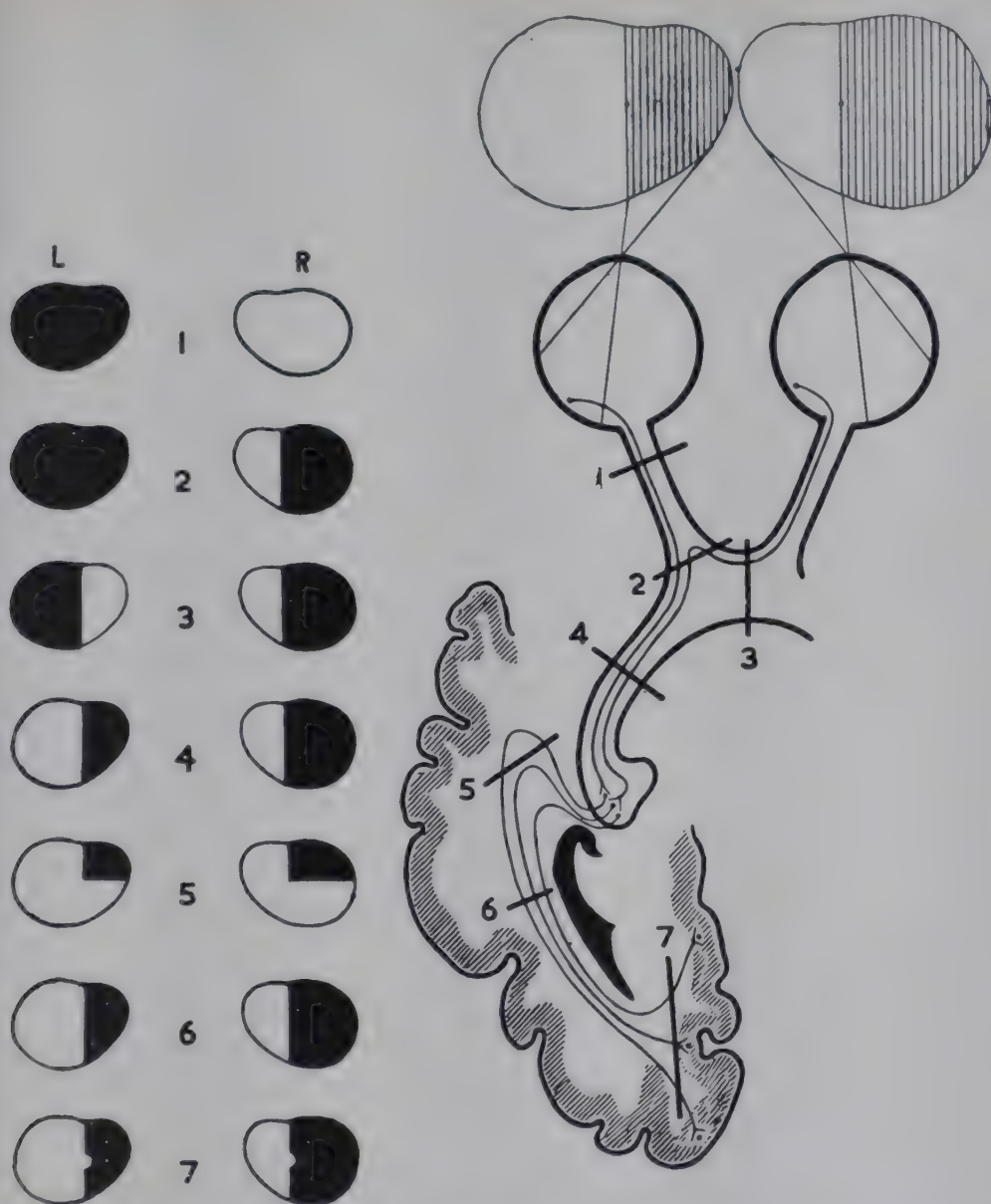


FIG. 269. Diagram of the visual paths, showing sites of lesions and the corresponding field defects.

1. Lesion through optic nerve—ipsilateral blindness.
2. Lesion through proximal part of optic nerve—ipsilateral blindness with contralateral hemianopia.
3. Sagittal lesion of chiasma—bitemporal hemianopia.
4. Lesion of optic tract—homonymous hemianopia.
5. Lesion of temporal lobe—quadrantic homonymous defect.
6. Lesion of optic radiations—homonymous hemianopia (sometimes sparing the macula).
7. Lesion of occipital lobe—homonymous hemianopia (usually sparing the macula).

in the occipital lobe or optic radiations (Fig. 269, 6, 7). The chief causes are injury by falls on the back of the head or gun-shot wounds, cerebral tumour, or cerebral softening due to disease of the blood vessels. In gun-shot wounds both occipital lobes are not infrequently injured; there is usually unconsciousness from concussion at first and the hemianopia becomes manifest with the gradual recovery. If both lobes are extensively injured there is complete blindness; often, however, some portion of the cortex of one or other calcarine fissure escapes, and in these cases some measure of central vision is regained.

In less extensive injury the hemianopic symptoms may gradually improve. The first sign of improvement is the perception of the movement of objects in the affected field without recognition of their nature and details. The onset of hemianopia due to disease of the cortex is more gradual, and careful investigation with the perimeter shows that the colour fields are often lost before the field for white light, although this is always contracted. This *hemiachromatopsia* is itself of gradual onset. In cortical and sub-cortical lesions the pupillary reactions are normal (p. 36), and the fundi reveal no ophthalmoscopic changes, except in the case of tumours which may be associated with bilateral papilloedema. Cortical lesions are liable to be accompanied by word-blindness, usually due to involvement of the angular gyrus. When the lesion is in the posterior part of the internal capsule hemianæsthesia, with or without hemiplegia, is likely to be present.

Rare cases of homonymous *quadrantanopia* have been reported, in which corresponding quadrants of each field—the upper or lower half of one temporal, and the upper or lower half of the other nasal—have been lost. These may be caused by cortical or sub-cortical partial lesions of one occipital lobe, destruction of the part above the calcarine fissure leading to loss of the lower quadrants and *vice versa* (Figs. 31 and 32). A similar quadrantic defect occurs in lesions of the temporal lobe owing to the fact that a ventral band of the optic radiations passes first forwards and then backwards in the temporal lobe in its course from the lateral geniculate body to the occipital lobe (Fig. 269, 5). Partial hemianopia of a quadrantic type is then commoner than the typical homonymous defect, usually greater on the side of the lesion. Subjective sensations of smell occur in some of these cases, due to the involvement of the uncinate process of the hippocampal gyrus.

Lesions of the Optic Tract. In these cases, since the afferent pupillary fibres part company with the visual fibres before the latter enter the lateral geniculate body (p. 36), Wernicke's hemianopic pupil reaction should be present (p. 106); but this reaction is always difficult to elicit. More assistance in diagnosis is afforded by collateral symptoms. The proximity of the crus cerebri, third and other cranial nerves, leads to other complications in the pathological

picture (p. 542). The association of hemianopia with contralateral third nerve paralysis and ipsilateral hemiplegia suggests a tract lesion. As a rule the fixation point does not escape in tract hemianopia. Partial atrophy of both optic nerves manifests itself by pallor of the discs in these cases, preceded in cases of raised intracranial pressure by papilloedema. The lesion is usually syphilitic meningitis or a gumma, tuberculosis or tumour of the optic thalamus or temporo-sphenoidal lobe; softening and hæmorrhage are rare. It is important that the patient is subjectively unaware of his visual defect in lesions above the geniculate body, but is conscious of a hemianopic defect from geniculate or infra-geniculate causes.

Lesions of the Optic Chiasma. Bitemporal hemianopia is usually caused by tumours in the region of the sella turcica, pressure by a suprasellar aneurysm or by chronic arachnoiditis; these press upon the chiasma, so that the fibres going to the nasal halves of each retina are destroyed (Fig. 269, 3). Tumours of the pituitary body are most common; but suprasellar tumours, particularly cranio-pharyngiomata derived from Rathke's pharyngeal pouch and suprasellar meningiomata must be considered.

Enlargement of the pituitary body, whether from functional hyperplasia, adenoma, or malignant growth, leads to visual defects in about 80 per cent. of cases, due to pressure upon the chiasma which lies immediately above it (Fig. 270) and upon the inner sides of the optic tracts. The earliest visual symptoms may be a unilateral central scotoma simulating retrobulbar neuritis for one side is usually compressed before the other. This may be followed by homonymous hemianopia from pressure on one tract, or rarely by *altitudinal hemianopia*, i.e., loss of the upper or more rarely the lower halves of the fields from pressure upon the chiasma; early loss in the upper half of the field may be caused by intra- or extra-sellar tumours, early loss in the lower half suggests a suprasellar tumour. More commonly bitemporal hemiachromatopsia, passing into a complete hemianopia, supervenes. The field does not show the accurate delimitation characteristic of homonymous hemianopia, but gradually contracts from the temporal side inwards and from above downwards, finally involving the nasal field from below upwards and leading to complete blindness (Plate XVIII). Then, or at a much earlier stage, the vision of the other eye becomes affected in a similar manner. If the second eye becomes involved before vision is lost in the first, the fields show bitemporal hemianopia. Complete temporal hemianopia in one eye, for example, may be associated with temporal achromatopsia in the other; such a combination emphasizes the importance of charting the colour fields in all cases. Many patients have a homonymous hemianopia, due to pressure and traction on one optic tract. Variations in the type and progress of the visual defects are thus not uncommon.

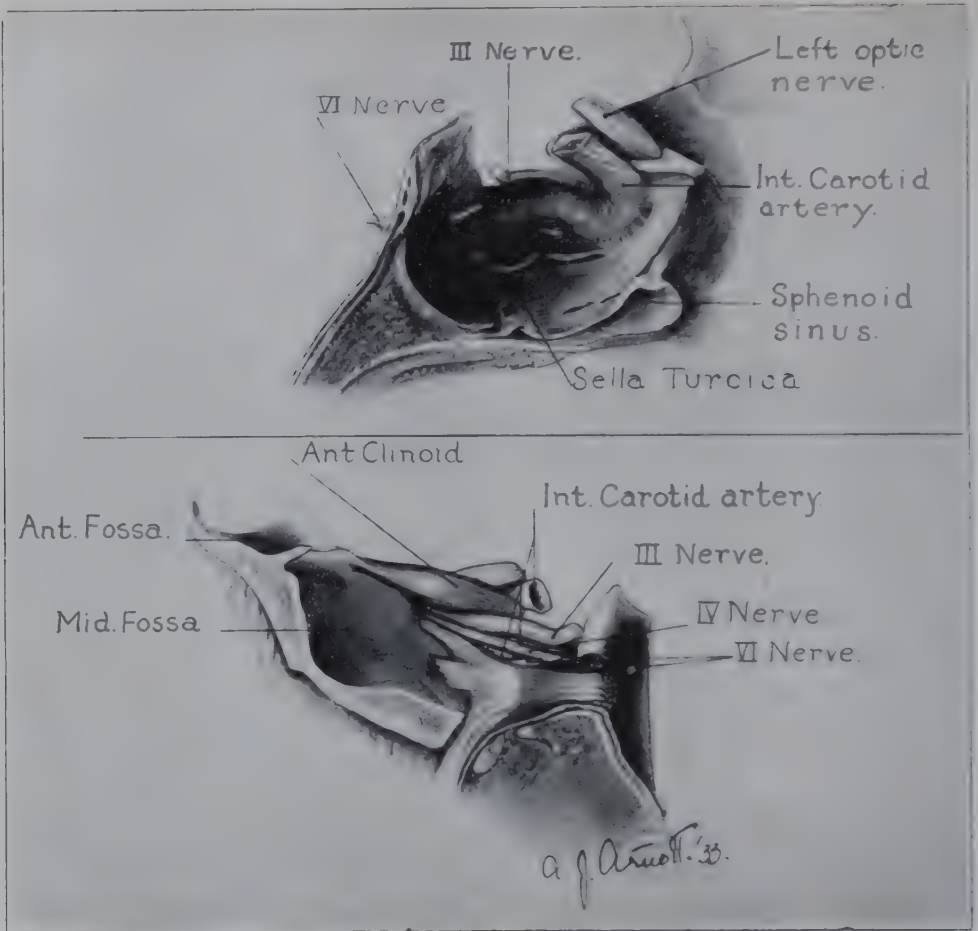


FIG. 270. The relations of the sella turcica in its medial and lateral aspects ; from a male with a chromophobe pituitary adenoma (Cairns).

A chronic *chiasmal arachnoiditis* of obscure origin may also cause bitemporal hemianopia due to compression of the chiasma by fibrous cicatricial bands. The same field defect has also resulted from antero-posterior injury to the chiasma in fracture of the base of the skull.

Binasal hemianopia is very rare. It necessitates two lesions, one on each side of the chiasma, destroying the fibres to the temporal halves of each retina while leaving the nasal fibres intact. It may be due to distension of the third ventricle, causing the optic nerves to be pressed downwards and outwards against the internal carotids, or to atheroma of the carotids or posterior communicating arteries.

Cases have been described in which there has been loss of half of the field in one eye and depression of vision progressing to blindness in the other. These are due to a lesion at the point where one optic nerve meets the chiasma so that the crossed fibres from the opposite side are involved as they loop forward into the nerve (Fig. 269, 2).

In all cases of chiasmal damage a careful survey of the central nervous system must be made as well as an enquiry into the function

of the pituitary. X-rays may provide valuable information, showing, for example, erosion of the sella, enlargement of the pituitary fossa or vascular calcification; simple radiography may be supplemented, if necessary, by encephalography and arteriography. If, as frequently occurs, vision progressively deteriorates, transfrontal exploration may be advisable; in many cases, particularly of pituitary tumours, the prognosis of operative removal, if undertaken in time, is reasonably good.

Amblyopia (ἀμβλύς, blunt) and **Amaurosis** (ἀμαυρός, dark) are the terms used for partial and complete loss of sight respectively in one or both eyes in the absence of ophthalmoscopic or other marked objective signs.

Unilateral amblyopia is usually either *congenital* or results from psychical suppression of the retinal image—*amblyopia ex anopsia*: these varieties are discussed elsewhere (p. 475). Unilateral amblyopia may be due to high refractive errors, a condition sometimes curable with suitable spectacles in early life if sufficient perseverance is exercised, but rarely in older people either because of the development of a true amblyopia from disuse or, more probably, defective receptivity of the higher centres. Unilateral amblyopia is also a symptom of retrobulbar neuritis (*q.v.*).

Bilateral amblyopia is found in the various forms of toxic amblyopia (*q.v.*). Bilateral amaurosis occurs in uræmia and in meningitis. Both amblyopia and amaurosis occur in hysteria.

Uræmic amaurosis occurs particularly in acute nephritis, especially complicating pregnancy or after scarlet fever, but is also found with chronic renal disease. The onset of blindness is sudden or rapid (8–24 hours); it is bilateral and complete. The fundi show no changes, unless, as in some cases, there is a coincident hypertensive retinopathy. Vision usually improves in 10–18 hours, and is fully restored in about 48 hours, especially if a lumbar puncture is done. In cases occurring during pregnancy there is usually eclampsia. In uræmic amaurosis the pupils are dilated, but generally react to light, showing that the lower centres are not affected. The condition is probably due to circulation of toxic material which acts upon the cells of the visual centres.

Amaurosis fugax is a term given to sudden temporary failure of sight usually due to circulatory failure. In its simplest form it occurs in normal people on rising suddenly from the sitting or recumbent posture when it is due to the effects of gravity, accompanied by slight giddiness and even faintness. It also occurs in aeroplane “black-out.” Transient blindness, seldom complete, occurs as a prodromal symptom of obstruction of the central artery of the retina, and is probably due to the occurrence of emboli or to the effects of changes in blood pressure associated with arterial disease. It has been met with in people with mild signs of Raynaud’s disease and most commonly in occlusion of the carotid artery. Temporary amblyopia also occurs in migraine (*vide infra*).

Hysterical amblyopia, as might be expected, exhibits protean manifestations. It may be unilateral, but is more commonly

bilateral. There is usually concentric contraction of the fields, with or without colour defects ; very characteristic is a spiral field which continually diminishes while it is being taken, so that it may be finally limited to the fixation point. The patients, however, get about perfectly well unaided, an impossibility in cases of genuine contracted fields (p. 328). Sometimes there are irritative symptoms—blepharospasm, blinking, lacrimation. The pupillary reactions are perfect, affording an invaluable objective diagnostic sign. Treatment must be directed to the psychogenic cause, but great care must be taken to eliminate organic disease before the diagnosis is finally accepted.

Scintillating Scotomata of various kinds occur in *migraine*. In typical migraine the patient feels unusually well before the attack. A positive scotoma appears in the field of vision ; while obscuring sight it has a peculiar shimmering character. It gradually increases in size until ultimately one-half of the field is clouded, the fixation point remaining relatively clear. In the dark field bright spots and rays of various colours are often seen, frequently arranged in zig-zags when they are called “fortification spectra” (*teichopsia*). Both half fields are usually affected, so that there is homonymous hemianopia. In other cases the whole field becomes clouded, but in spite of this the fixation point is usually seen momentarily, and then becomes obscured until the eyes are moved to a fresh spot. Vision usually clears in about a quarter of an hour, but the attack is soon followed by violent headache, generally intensified on the side of the head opposite the hemianopic field (*hemicrania*), and accompanied by nausea and even sickness (“bilious attack”). During the attack numbness in the mouth and tongue as well as slight aphasia are frequent as well as a copious secretion of urine of low specific gravity. Attacks occur periodically, but vary greatly in number and severity. In mild attacks, and especially as age advances, the scotoma may occur without the headache or the headache without scotoma.

Migraine is to be attributed to vasomotor changes in the brain. Vasodilatation, associated with a feeling of well-being, is followed by vasoconstriction, especially in the occipital lobes. It occurs chiefly in highly-strung people and may sometimes be accentuated by refractive errors ; some people—but they are few—have been cured by the wearing of suitable spectacles. A sedentary life with constipation and insufficient exercise conduces to the attacks. Rest, warmth, and sleep are the best measures to combat the attacks ; they can sometimes be warded off or alleviated by ergotamine tartrate.

Occasionally people who suffer from ordinary migraine have attacks in which, without any scotoma, the headache is followed by partial paralysis of the third nerve (*ophthalmoplegic migraine*) on the same side

as the hemicrania. Slight ptosis, diplopia, and sluggishness of the pupillary reactions continue for some hours and gradually disappear. The paresis is worse and persists longer with succeeding attacks, and has sometimes eventually become permanent. Probably most of these cases are not migrainous, but due to some organic nerve lesion such as pressure on the nerve by a distended artery ; some of the patients have died from subarachnoid hæmorrhage (p. 534).

Night-blindness occurs *par excellence* in pigmentary retinal dystrophy (*q.v.*) and in xerophthalmia (*q.v.*), but in rare cases it is a familial congenital affection. It is also found after exposure to bright sunlight in hot countries amongst patients who are debilitated by malnutrition or prolonged fasting ; the condition generally improves rapidly if the eyes are protected and the nutrition attended to. The affection is local, as is shown by the fact that covering one eye with a bandage during the day has been found to restore sight enough for the ensuing night, the unprotected eye remaining as bad as ever. Night-blindness is to be attributed to interference with the functions of the retinal rods, due to deficiency in visual purple. In xerophthalmia and the endemic cases the symptom is a manifestation of deficiency of fat-soluble vitamin A in the diet, and therefore cod-liver oil is specially indicated. It also occurs in diseases of the liver, especially cirrhosis, and may appear as a functional nervous disorder associated with other symptoms of neurosis or malingering.

Coloured Vision (Chromatopsia) is a rare symptom. *Erythropsia* (red vision) occurs particularly after cataract extraction if the eyes are exposed to bright light. Objects look red, but the visual acuity is not affected, and no permanent damage results. Patients should be warned of the possibility of erythropsia, as it is somewhat alarming and suggestive of hæmorrhage. It is met with also in snow-blindness. Chromatopsia also occurs in some cases during the resolution of optic neuritis when the ensuing atrophy is not complete. In normal people black print will sometimes suddenly turn deep red owing to strong lateral light entering the eye through the sclera.

Colour Blindness or Achromatopsia may be congenital or acquired. *Acquired colour blindness*, partial as in cases with relative scotomata or complete as in disease of the optic nerve, has been referred to incidentally in discussing the various disorders of the eye in which it occurs. In most diseases of the retina and choroid, changes in colour perception affect mostly the blue end of the spectrum. Slight diminution in acuity of perception of these rays is caused normally, owing to their physical absorption, by the increase of amber pigment in the nucleus of the lens (*blue blindness*), and this may be abnormally great in sclerosing lenses (black cataract) ; it has been said to affect the pictures of artists in their old age.

Congenital colour blindness occurs in two chief forms, total and partial. The former is very rare and is generally associated with nystagmus and a central scotoma. All colours appear grey, of different brightness. The spectrum appears as a grey band like the normal scotopic spectrum (p. 28), and like it with the maximum

brightness at 510 m μ . It is probable that total colour blindness is caused by a central defect.

The partial form is seldom discovered unless special tests are made since the subjects compensate for their defect by attention to shade and texture, combined with experience. Gross cases occur in 3 to 4 per cent. of males, but are rare in females (0.4 per cent.) ; slighter cases are more common in males. It is an inherited condition, being transmitted through the female who is usually unaffected, and is probably due to the absence of one of the photopigments normally found in the foveal cones. In most cases reds and greens are confused, so that the defect is a source of danger in certain occupations, such as engine-drivers and sailors. The red-green cases fall into two chief groups, *protanopes* and *deutanopes*. For the former the red end of the spectrum is much less bright than for normal people and is often actually shortened ; in deutanopes the green sensation is defective. These groups are said to have *dichromatic* vision. In both groups the defects may not be complete and these cases are called *protanomalous* and *deuteranomalous* respectively. It is clear that theoretically there might be other cases of colour blindness due to absence of the blue sensation, and such have been described, but are very rare (*tritanopes*).

There are two objects in testing for colour blindness : (1) the exact nature of the defect ; (2) whether the subject is likely to be a source of danger to the community. The first is an exhaustive investigation involving stringent tests with a pure spectrum. In testing for danger, it is obvious that the names given to the colours are of value, for if a man repeatedly calls red green or *vice versa* he is unsuited to be an engine-driver or look-out man on a ship. Whatever the object in view, several tests should be employed ; for the spectrum tests the student must be referred to special monographs on colour vision. The following are the other chief tests.

(1) *The Lantern Test*. The subject names various colours shown by a lantern, and is judged by the mistakes he makes. Much here depends upon the size of the apertures of the lantern (i.e., the size of the retinal areas stimulated) and the nature and intensity of the light source. Many lanterns are worse than useless. Edridge-Green's lantern is efficient if used by an expert.

(2) *Holmgren's Wools*. These consist of a selection of skeins of coloured wools from which the candidate is required to make a series of colour-matches. This test has been much criticized, but if properly carried out gross defects of colour vision are easily recognized and an expert will be put on his guard in almost every case of even minor defect.

Test I consists in presenting to the candidate a pale green sample and telling him to select from the heap of wools all those which seem to correspond in colour. If he is colour defective he will probably select several of the "confusion colours"—greys, buffs, straw colour, etc.—as well as greens. He is next given a rose colour (II) : if he matches this with blues or violets he is red-blind ; if with greys or greens he is green-blind. He is then given a bright red skein (III) : if he is red-blind he will choose dark greens and browns, if green-blind pale greens and browns. IV is a purple skein : if the candidate is colour defective he will probably select any shade of blue or green, also pinks and

greys. V is a yellow skein : the colour-defective candidate will probably select greenish-yellows, light yellow-greens, fawns and pinks. In blue-blindness purples, red and orange are confused in test II.

(3) *Isochromatic Charts*. These consist of coloured lithographic plates in which bold numerals are represented in dots of various tints set amid dots of the same size but of tints which are most readily confused with those of the figures by colour-defective people. Normal trichromats can easily read the numbers, some of which are indistinguishable to the various types of colour-defective while tests are included in which the numbers can be read by the colour-defective, but not by the normal sighted. Stilling's original tests have now been largely replaced by the Japanese test of Ishihara, while good alternatives are the Swedish Boström test or the American H-R-R test.

(4) *Nagel's Anomaloscope*. This is an instrument in which on looking down a telescope a bright disc is seen, divided into two halves by a horizontal line. One half is illuminated by light of the sodium line of the spectrum (yellow), and this has to be matched by a mixture of red (lithium line) and green (thallium line) in the other half. By turning screws the relative amounts of red and green in the mixture and the brightness can be varied.

Defects of colour vision have led to much acrimonious discussion. Their detection may be easy, but is often difficult and no single test is infallible.

Word-blindness occurs as a not very uncommon congenital anomaly, due to defects in the association areas of the brain, which often runs in families ; it affects 0.1 per cent. of primary school children, being much commoner in boys than girls. Owing to backwardness in learning to read the children are often brought to the ophthalmic surgeon because a visual defect is suspected. In spite of normal fundi and often normal acuity of vision, the patients fail to recognize printed or written words. The auditory memory of words is unimpaired, and generally numerals and music can be read. Hence the patients learn well orally and are good at arithmetic. They are often quite intelligent and may be wrongly punished for inattention and stupidity. The defect is not necessarily complete, and much improvement can be obtained by careful individual tuition and perseverance. As an acquired condition (*aphasia*) after a central lesion, it is rare.

Malingering. Cases occasionally occur of people who hope to gain some advantage by pretending to be visually defective. It is rare for complete blindness to be assumed, and such cases can only be detected by constant watching of the person's behaviour. When one eye is said to be blind, in spite of absence of sufficient objective evidence to account for the condition, the demonstration of malingering resolves itself into a contest of wits between the surgeon and the individual. Many tests have been devised, and several should be employed in each case.

(1) A low concave or convex glass (0.25 D) is placed before the "blind" eye, and a high convex (+ 10 D) before the "good" eye, and the examinee is told to read the distant types. If he succeeds malingering is proved.

(2) A prism is placed base downwards before the "good" eye and the examinee is told to look at a light. If he admits to seeing two lights malingering is proved.

(3) The surgeon stands behind the patient and covers the "blind" eye with his hand, at the same time holding a prism of 10 degrees base down before the "good" eye in such a manner that the edge of the prism passes horizontally across the centre of the pupil; uniocular diplopia results. The surgeon then simultaneously removes his hand from the "blind" eye and shifts the prism upwards so that the whole pupil is covered by it. If the examinee still admits to seeing two lights malingering is proved.

(4) While the examinee looks at a light a prism of 10 degrees is placed base outwards before the "blind" eye. If the eye moves inwards in order to eliminate diplopia it is not blind.

(5) Snellen's coloured types may be employed. The letters are printed in green and red. If a red glass is placed before the "good" eye, and the patient reads all the letters, the other eye is not blind, for the eye looking through the red glass can only see the red letters. Care must be taken in this test that the red glass cuts off all the rays from the green letters as tested by the surgeon's own vision.

CHAPTER 25

INTRA-OCULAR TUMOURS

INTRA-OCULAR tumours are rare, but of great importance, since they are usually malignant and endanger the life of the patient.

TUMOURS OF THE UVEAL TRACT

The common primary malignant tumour of the uveal tract is derived from the sheaths of Schwann and is thus ectodermal ; it is best, therefore, to refer to these tumours as *malignant melanomata*.

Tumours of the Iris. It is not uncommon to see irides with dark brown spots (melanomata), due to congenital aggregations of

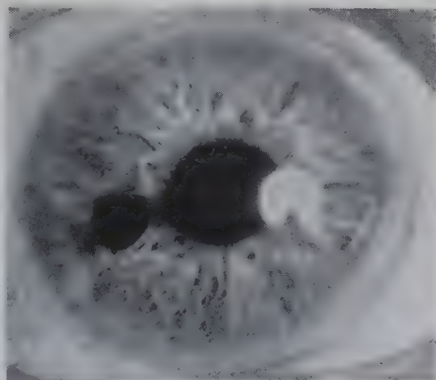


FIG. 271. Malignant melanoma of the iris.
(Note the deformation of the pupil.)

pigment cells. As a rule these are benign *nævi*, but occasionally they take on malignant proliferation. Any increase of size must be watched with suspicion.

Malignant melanoma is the only neoplasm of importance met with in the iris but is rare. Composed of pigmented or unpigmented spindle-shaped or round cells, it occurs as an isolated nodule which grows rapidly (Fig. 271), and if untreated penetrates the corneo-sclera and perforates the globe ; it gives rise to fatal metastases. Diagnosis from a granulomatous lesion depends on the absence of inflammation and the density of pigmentation, but the occasional absence of pigmentation may give rise to difficulties.

Treatment. The growth should be watched for a short time, preferably by repeated photography and if found to increase in size should be removed by a wide iridectomy if this is feasible. If microscopic investigation shows that the growth is malignant and has not been completely removed or shows signs of recurrence, it is

safest to excise the eye. If completely removed the prognosis is good.

Malignant Melanoma of the Choroid arises from the outer layers of this tissue. It forms at first a lens-shaped mass, raising the retina over it. As it grows tension is thrown upon the elastic membrane of Bruch, which finally ruptures; the tumour then proliferates through the opening to form a globular mass in the subretinal space, separated from the part in the choroid by a narrow "neck" (Fig. 272). The retina remains in contact with the tumour at the summit, but is detached from the choroid at the sides, the intervening space being filled with exudative fluid. The growth may be in any situation, and the fluid may sink down to the lowest part

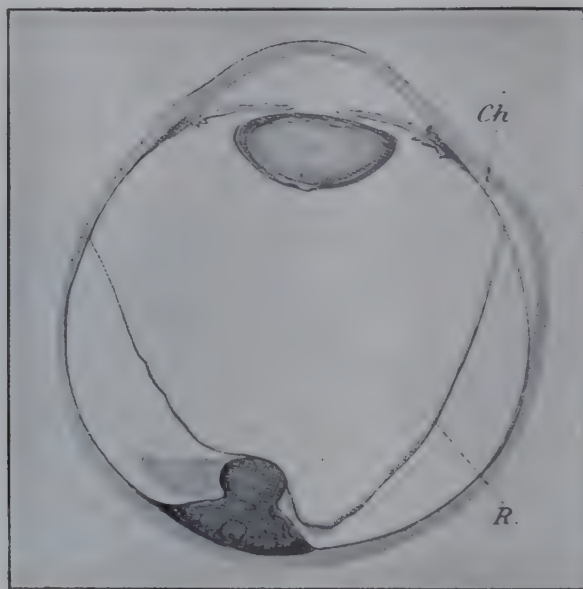


FIG. 272. Section of malignant melanoma of the choroid, showing the typical mushroom shape. *Ch.*, pars plana of ciliary body, continuous posteriorly with the choroid, behind *R.*, retina.

of the eye, forming there a detachment isolated from that over the tumour, but with continuing growth the retina becomes more and more detached, until no part remains *in situ*; the nutrition of the lens then suffers, so that it becomes opaque. The tumour may fill the globe before perforating the sclera, or this may occur relatively early along the perivascular spaces of the vortex veins or ciliary vessels. The orbital tissues then become infiltrated. The lymph nodes are not affected, but metastases occur abundantly in the liver and elsewhere.

The growth is usually pigmented but is occasionally unpigmented, a distinction which is relatively unimportant; metastases from melanotic growths are often unpigmented. The pigment is chiefly melanin, but hæmatogenous pigmentation occurs after hæmorrhages.

The cells are usually spindle-shaped ; they may be cylindrical or palisade-like, arranged in columns or around blood vessels, or even endothelial in appearance ; most tumours are mixed-celled. Silver staining reveals a variable amount of argyrophil "reticulin" fibres, generally more numerous in spindle-celled sarcomata. There is evidence that those with the most reticulin are the least malignant.

Flat Malignant Melanoma of the Choroid. In rare cases the choroid becomes widely infiltrated so that a uniform thickening results, with a shallow "detachment" of the retina. Some of these may be endotheliomatous growths, spreading along the lymphatic spaces of the choroid in the same manner as secondary carcinoma (*q.v.*).

The *clinical course* of malignant melanoma of the choroid is commonly divided into four stages : (1) the quiescent stage ; (2) the glaucomatous stage ; (3) the stage of extra-ocular extension ; (4) the stage of metastasis. This is probably the typical chronological order of events, but secondary glaucoma may arise at an early stage or be delayed until after extra-ocular extension has taken place, and metastases may occur at any stage.

The cause of the glaucoma is obscure : in some cases it is due to the lens and iris being forced forwards, so that the angle of the anterior chamber becomes blocked or it may be infiltrated with neoplastic cells. In other cases, particularly those of early onset, obstruction to the venous outflow from the eye is a possible explanation, the tumour being in some instances so situated as to press upon a vortex vein.

The tumour usually occurs in adults between forty and sixty. It is always primary, single, and unilateral. The earliest patients to seek advice are those in whom the tumour is near the macula, since vision is then most strikingly affected. In the periphery the tumour has usually attained a considerable size, and the patient may apply for treatment for relief of the pain of glaucoma.

It is of the utmost importance that the cause of the detachment of the retina should be diagnosed in these cases. If the tension is raised, a growth may be diagnosed almost with certainty. A simple detachment shows numerous more or less parallel folds, and undulations can be seen to travel over the surface when the eye moves. The detachment at the summit of a tumour is usually rounded and fixed, though in the surrounding parts it may show all the signs of a "simple" detachment. Patches of pigment upon the rounded part support the diagnosis of a tumour, but pigmentary disturbance, more particularly at the periphery, is not uncommon in simple detachment. A simple detachment of the non-exudative type probably always has a hole or tear in the retina somewhere ; if it can be found it is the most positive evidence that a growth is probably not present. Rarely a system of blood vessels having an entirely different mode of distribution from the retinal vessels can be made

out between the latter ; this is the most positive evidence of a growth, but it is only occasionally seen. A very small, round detachment in the macular region or upper part of the globe is almost certain to be due to a tumour of the choroid. If the detachment is anterior, transillumination (p. 109) will afford assistance in diagnosis ; a simple detachment is transparent, a choroidal growth opaque.

Diagnosis may be extremely difficult if the patient is first seen when glaucoma has already supervened. Dependence must then be placed largely upon the history. Defective vision may have been noticed, but the premonitory halos of glaucoma have been absent, and vision has gone from bad to worse without remissions. One eye only is involved ; the other may be normal, or at least not of the glaucomatous type with a narrow angle, and the field of vision in this eye will show no typical glaucomatous defects. The affected eye will probably have no perception of light, so that if any doubt remains it should be excised. The frequency of the occurrence of a neoplasm in such eyes should make this an invariable rule.

In the diagnosis, two rare simple tumours must be kept in mind, particularly in the early stages. A *choroidal nævus* appears as a bluish patch with somewhat feathered edges, usually about the size of the optic disc and situated near the posterior pole of the eye. It is congenital and symptomless but, like nævi elsewhere, may occasionally assume malignant characteristics.

A *cavernous hæmangioma* of the choroid, another rare tumour of congenital origin and of exceedingly slow growth, is also usually situated near the disc. It has a greyish hue and indefinite margins and often causes a retinal detachment.

Treatment. The eye should preferably be excised as soon as possible after arriving at the diagnosis. Although such growths rarely travel down the nerve, it is wise to cut it as far back as possible. If the growth has already burst through the globe, the orbit should be exenterated and, in addition, irradiated with X-rays or radium. When the tumour is small and the affected eye is the only seeing eye, destruction by diathermy, light-coagulation or suturing cobalt discs to the sclera over the site of the neoplasm (p. 370) may be considered.

The disease is invariably fatal, usually within five years, if not eradicated by operation, but metastases may be delayed for ten years or more. Prognosis is fair if the tumour is small and entirely intra-ocular, especially if it contains much reticulin.

Malignant Melanoma of the Ciliary Body is fundamentally of the same nature and gives rise to the same symptoms as the corresponding neoplasm of the choroid, the differences being only those dependent upon the anatomical disposition of the parts (Fig. 273). Thus the retina, being here more adherent to the underlying uvea and being reduced to a double layer of epithelial cells, is not detached until the growth has spread to the choroid. The tumour may attain a considerable size before it causes symptoms,

which are then referable to displacement or distortion of the lens and interference with the ciliary muscle. The ciliary circulation is impeded, and conspicuous dilatation of one or two anterior perforating ciliary vessels should always arouse suspicion. The growth may invade the angle of the anterior chamber when it then has the appearance of an iridodialysis, a dark crescent showing at the root of the iris ; that it is not an iridodialysis is shown by the fact that no reflex can be obtained through it on illuminating with the ophthalmoscope and from the absence of a history of a blow. In an unpigmented tumour the crescent may be yellowish, but vessels will usually be visible upon the surface and these render the diagnosis easy. The growth may be visible by oblique illumination with

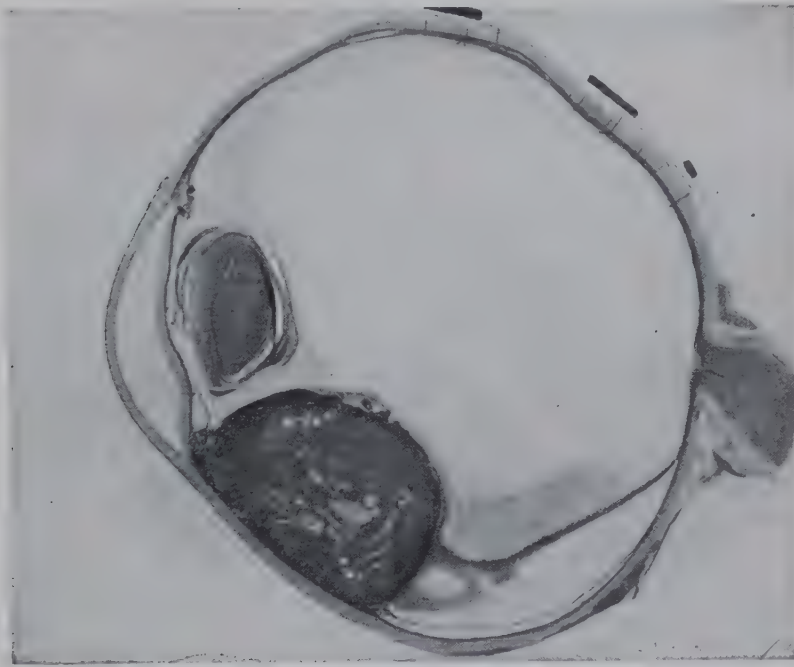


FIG. 273. Malignant melanoma of ciliary body (Coppez).

a widely dilated pupil and by gonioscopy, and is opaque to transillumination. Occasionally it takes on a *ring* or *annular* distribution, extensively infiltrating the ciliary region.

Malignant melanoma of the ciliary body is less common than that of the choroid ; the treatment and prognosis are the same.

Epithelial hyperplasia of the ciliary processes is not uncommon in old eyes. Rarely *malignant epitheliomata* occur ; also growths resembling embryonic retina (*diktyoma*). They cause the same clinical signs as malignant melanoma.

Secondary Carcinoma of the Choroid occurs as a metastatic growth in cases of cancer, particularly of the breast and alimentary tract. There is obscuration of vision, and ophthalmoscopic examina-

tion reveals a widespread shallow elevation of the retina, usually at the posterior pole. The disease is nearly always bilateral, and as it is usually only one of many metastatic deposits and the patient is generally in the stage of general carcinomatosis, excision of the eye is not usually indicated. These metastases, however, are radio-sensitive and treatment by radiation will often effect sufficient improvement to maintain some vision and prevent the occurrence of pain until death supervenes.

TUMOURS OF THE RETINA

Retinoblastoma used to be commonly known as “glioma” retinae; malignant proliferations of neuroglia such as occur in the brain and optic nerve, are very rare in the retina. The usual malignant growth of the retina is due to proliferation of neural cells which have failed to evolve normally, and is better termed retinoblastoma.

The tumour is confined to infants and is probably always congenital, although it may remain quiescent or pass unnoticed until the fifth or sixth year or even later. The disease is rare; the second eye is affected, independently and not by metastasis or continuity *via* the chiasma, in about one-fourth of the cases, but frequently the growth cannot be recognized even on careful examination until after months or even years. Several children of the same family are sometimes affected.

The child is brought to the surgeon on account of a peculiar yellow reflex from the pupil, sometimes called “amaurotic cat’s eye.” If left untreated retinoblastoma runs through the same stages as melanoma of the choroid—(1) the quiescent stage, lasting from six months to a year; (2) the glaucomatous stage; (3) the stage of extra-ocular extension; (4) the stage of metastasis. The second stage results in enlargement of the globe, with apparent or real proptosis. Pain is severe during this stage, but is relieved when the tumour bursts through the sclera, an event which usually occurs at the limbus and is followed by rapid fungation. Metastasis first occurs in the pre-auricular and neighbouring lymph nodes, later in the cranial and other bones. Direct extension by continuity to the optic nerve (which is early affected) and brain is more common, and metastases in other organs, usually the liver, are relatively rare.

The growth consists chiefly of small round cells with large nuclei resembling the cells of the nuclear layers of the retina; many of these stain badly, showing that they are undergoing necrosis (Fig. 274). Among them may be found rosette-like formations of cells resembling the rods and cones.

Retinoblastoma is invariably multiple. When noticed very early, as may occur in the second eye, a larger mass is seen surrounded by numerous punctate satellites. Microscopically, minute deposits are seen scattered in various situations throughout the globe. It may

grow principally outwards, separating the retina from the choroid ("*glioma exophytum*"), or inwards towards the vitreous ("*glioma endophytum*"); between the two there is no fundamental distinction, but the ophthalmoscopic appearances differ. In the former the condition resembles a detachment of the retina; in the latter poly-poid masses, sometimes with hæmorrhages on the surface, may be seen spreading into the vitreous.

Several conditions occurring in children may give rise to similar signs, and cause great difficulty in diagnosis. These have been grouped together under the term "*pseudo-glioma*." The chief are (1) inflammatory



FIG. 274. Section of retinoblastoma. Note the rings of deeply stained cells surrounding blood vessels; also the infiltration of the anterior part of the optic nerve.

deposits in the vitreous, with or without detachment of the retina following a plastic cyclitis or choroiditis; (2) tuberculosis of the choroid, especially the confluent type; (3) toxocara infestation; (4) congenital defects, due to persistence of part of the fibro-vascular sheath at the back of the lens; and (5) retrolental fibroplasia. In all cases atropine should be instilled and both eyes should be thoroughly examined ophthalmoscopically, under general anæsthesia if necessary. The tension may then be satisfactorily tested and may afford useful information which cannot be obtained without an anæsthetic. Raised tension is in favour of retinoblastoma, lowered, of pseudo-glioma. Even when every precaution is taken, in some cases it is impossible to be certain of the

diagnosis ; considering that the life of the patient is at stake and that the eye is in any case useless as an organ of sight, these should be treated as malignant.

Treatment. The treatment is excision of the eye at the earliest possible moment. The optic nerve should be cut long, and the cut end invariably submitted to microscopical examination. If there is any doubt of extension of the disease to the conjunctiva or orbital tissues exenteration of the orbit is indicated. When the diagnosis is doubtful the eye should be removed, for in inflammatory pseudo-glioma the eye is destined to shrink and become unsightly. In no case should both eyes be removed at the same operation, but if one is proved by microscopical examination to contain retinoblastoma and the other contains similar nodules, it is justifiable to treat these by destruction by diathermy or irradiation provided they do not occupy an area of more than one-third of the retina.

In treatment by radiation the sources used are radium applicators, radioactive cobalt discs or tantalum wire stitched to the sclera over the site of the nodule ; the object is to deliver a dose of 4,000 r to the summit of the tumour in one week. Late sequelæ of irradiation are thin greyish exudates at the macula appearing eighteen months after treatment, and posterior cortical lens opacities becoming evident after a period varying from nine months to eight years. Alternatively, X-rays may be used with an apparatus to confine the radiation to the posterior segment of the globe and thus avoid damage to the lens.

The prognosis of retinoblastoma, if untreated, is always bad ; the patient invariably dies. The prognosis is fair if the eye is removed before extra-ocular extension has occurred. In the absence of disease of the second eye the patient may be regarded as out of danger if there is no recurrence in the orbit within three years, but the remaining eye should be carefully examined under atropine at frequent intervals for a much longer period. There are several cases on record of survival after removal of both eyes. Owing to its familial tendency the eyes of subsequent siblings or descendants should be carefully watched during infancy and childhood.

CHAPTER 26

INJURIES TO THE EYE

THE eye is protected from direct injury by the lids and the projecting margins of the orbit. Nevertheless, it is not exempt from foreign bodies, the action of caustics, contusions and wounds.

EXTRA-OCULAR FOREIGN BODIES

Foreign Bodies, which are usually small—particles of coal dust, emery, steel, etc.—may pitch upon the conjunctiva or upon the cornea. In the former case they cause sudden discomfort and reflex blinking. The foreign body sticks to the palpebral conjunctiva and is liable to be dragged across the cornea, which it excoriates. It may be washed by tears towards the inner canthus, and so into the nasal duct; more frequently it becomes lodged at about the middle of the upper sulcus subtarsalis where it is most likely to irritate the cornea, or in the upper fornix, or it may occasionally become embedded in the bulbar conjunctiva. Quite large foreign bodies, such as a grain of corn, may be retained for a long time in the upper fornix and give rise to much irritation and some discharge; they may be overlooked unless the upper lid is everted. They are generally embedded in a mass of granulation tissue, which may simulate the cockscomb type of tuberculosis (p. 172). The wing-cases of insects and the husks of seeds may adhere by their concave surfaces to the cornea, usually at the limbus, for several weeks.

Particles of steel and emery are very liable to fly straight onto the cornea and penetrate into the epithelium or substantia propria. Larger particles of steel, or less commonly stone, glass, etc., may perforate the globe (p. 390). When situated in the cornea they cause great pain and irritation. The pupil is often constricted. If allowed to remain they expose the cornea to the dangers of infection by organisms in the conjunctival sac with resultant ulceration which may lead to the extrusion of the foreign body in the slough. The ulcer thus formed may heal, but if virulent organisms are present a spreading ulcer, with or without hypopyon, may develop.

It is not always easy to discover a foreign body upon the cornea. If situated eccentrically thereon, a leash of dilated conjunctival blood vessels will point in its direction. In case of doubt the eye should be anaesthetized and the cornea thoroughly examined under oblique illumination with a loupe. The use of fluorescein will sometimes, but not always, reveal the position. In some cases the slit-lamp is of great assistance in determining the nature and position of the foreign body,

for by its use the depth of an embedded foreign body can be estimated by the length of the shadow which it casts (Fig. 275).

Treatment. Foreign bodies must be removed as soon as possible. If lying loosely in the lower fornix they are easily removed with a clean swab or handkerchief after everting the lower lid. If not found in this position the upper lid should be everted (p. 98); the particle will generally be found in the sulcus subtarsalis and can be removed in the same manner. If it is still not seen the upper fornix should be brought into view (p. 98) and the particle removed. In case of difficulty, the previous anæsthetization of the conjunctival sac will materially assist.

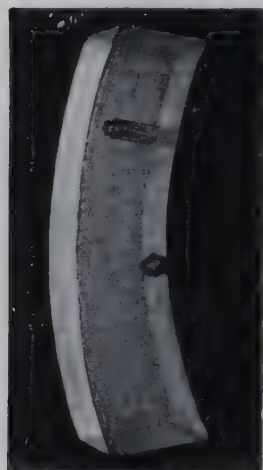


FIG. 275. The location of foreign bodies in the cornea by the slit-lamp. The beam of light comes from the left. The upper foreign body is in the middle of the stroma and throws a long shadow. The lower foreign body lies on Descemet's membrane.

If the foreign body is embedded in the bulbar conjunctiva it should be picked out by a needle after topical anæsthesia. If a discission needle is not at hand a darning needle may be used; it should first be passed through a flame, so as to sterilize it. This little operation is performed in the same manner as removal of foreign bodies from the cornea, but it may be necessary to snip off the small piece of conjunctiva containing the foreign body with scissors.

Removal of foreign bodies from the cornea is effected as follows:

The eye is anæsthetized and the patient seated in a chair. The surgeon holds the lids apart with the first and second fingers of his left hand, pressing slightly backwards so as to steady the globe. A light is focused upon the cornea, the patient being told to look in the direction which affords the best view of the particle. An attempt may first be made to remove the foreign body by touching it with a slip of clean blotting-paper which exercises a capillary attraction. If this fails a sterilized spud (Fig. 276) is used. Only if this also fails after repeated efforts should a needle be used, preferably a discission needle. The greatest care should be exercised not to scrape the epithelium more than is absolutely necessary. Emery and steel particles leave behind them a little ring of brown stain which should be scraped off if this can be done without too much trauma.



FIG. 276. Spud for removing foreign bodies from the cornea.

If there is any sign of ulceration or a greyish infiltration around the abrasion, and if the probability of acute (closed-angle) glaucoma can be excluded, a drop of 1 per cent. atropine should be instilled, warning being given that the sight will be misty for a few days. If the angle of the anterior chamber is suspiciously narrow atropine

should be avoided and the eye should be examined daily for a time. Atropine should not be used as a routine measure : it is generally unnecessary, and it involves prolonged visual disability. In every case an antibiotic ointment should be inserted and the eye kept bandaged for a day. If ulceration occurs it is treated in the appropriate manner (p. 192). Special attention should be given to particles of stone which show a greater tendency than metal to cause ulceration, probably because metallic particles are often hot and therefore sterile when they enter the eye.

Occasionally sharp steel and other particles penetrate deeply into the cornea, without, however, perforating. The efforts made to remove them may push them in still deeper or even into the anterior chamber. When such an accident is feared, special precautions must be adopted. If the particle is magnetizable and a large magnet is available, magnetic removal should be tried (p. 396), but it is usually necessary to incise the cornea overlying the foreign body. This method may fail, particularly if the particle is small. If the foreign body escapes into the anterior chamber it must be removed by other methods (p. 396).

Fragments of aniline pencil in the eye cause much irritation and a very unsightly staining and may lead to ulceration and necrosis. The eye should be treated with glycerin drops (p. 559), since this substance is a solvent of aniline violet.

Prophylactic Measures. Foreign bodies in the eye are extremely common in industrial workers, especially in grinding tools, lathe work, or hammering on a chisel with a mushroomed head. Apart from the danger to the sight of the worker, they are a source of great economic loss from expenditure of time and compensation. In addition to the banning of tools with overhanging edges, the fitting of guards on machines for grinding and other available preventive measures, such accidents could in most cases be entirely prevented by the use of goggles, but it is often impracticable to enforce this measure among workmen. Every attempt should be made by the provision of comfortable goggles and by educative means, such as "Safety First" notices and lectures by welfare officials, to point out the dangers of failure to protect the eyes in this way.

BURNS AND CHEMICAL INJURIES

Burns by hot water or steam, hot ashes, exploding powder or molten metal, and injuries by caustics such as lime usually from fresh mortar or whitewash, or strong acids and alkalis may involve considerable damage either by injuring the cornea or by producing symblepharon. Strong ammonia is particularly harmful, causing necrosis of the cornea ; acids (hydrochloric, sulphuric) much less so. Immediately after the accident there is intense conjunctivitis and chemosis, but the cornea often looks clear ; in this state it is difficult to be certain of the severity of the injury. A drop of

fluorescein solution will reveal the extent of the area denuded of epithelium. Prognosis should therefore be guarded, care being taken to impress upon the patient the gravity of the injury and the necessity for supervision. In the worst cases the cornea is dull or opaque. In the succeeding days an eschar forms and is thrown off ; this is followed by granulation of the injured conjunctiva and frequently by ulceration of the cornea. The corneal condition should be treated as an ulcer (p. 192). In severe lime burns the whole cornea may be destroyed ; perforation takes place, and the eye shrinks. In less severe cases a dense leucoma forms, porcelain-like in lime burns, and sight is lost. The chief danger derived from the condition of the conjunctiva is that of adhesion of the lid to the globe. It is most likely to occur with the lower lid where the lower fornix is obliterated by organization of the granulation tissue. The symblepharon thus produced impedes the movements of the globe and may even interfere with its nutrition. Every precaution must be adopted to prevent its occurrence.

Treatment. In injury by caustics the excess of deleterious material must be removed at the earliest possible moment. Acids may be neutralized by dilute alkalis (lotio sodii bicarbonatis, 3 per cent.) and alkalis by weak acids (lotio acidi borici) or milk. In no case, however, should there be any delay in obtaining these solutions and if they are not immediately at hand, the eye should be copiously irrigated at once with water. Particles of lime must be perseveringly picked out with forceps, after the previous instillation of pantocaine : irrigation with 10 per cent. neutral ammonium tartrate or preferably a solution of the sodium salt of ethylene-diamine tetra-acetic acid (Appendix I) diminishes scarring in lime burns. An antibiotic ointment should be instilled. A potent agent in reducing the inflammatory reaction and preventing the excess of granulation tissue which determines the development of symblepharon, is a corticosteroid applied topically as drops or ointment (Chapter 14).

Symblepharon may occasionally be prevented by sweeping a glass rod well coated with a lubricant round the upper and lower fornices, so that they are well packed with ointment, a procedure which should be repeated several times each day according to the severity of the case. This, however, is by no means invariably effective and, if the fornix is denuded of epithelium, it may be impossible to prevent the development of adhesions. In simple (traumatic) cases a graft of mucous membrane from the mouth may be an effective method of treatment, but in diseases such as pemphigus the graft is usually involved in the pathological process. In such severe cases, an alternative in a quiet period is to open up the fornices freely cutting the adhesions and allowing epithelial healing to occur under a large retained scleral contact shell. If the cornea is covered by vascularized scar tissue, this may often be peeled off leaving a relatively clear surface which may be retained under the contact shell for a considerable time.

Poison gases used in warfare include lacrimatory gases, phosgene, mustard gas, arsenicals, and other agents.

The *lacrimatory gases* include K.S.K. (ethyliodoacetate), B.B.C (bromobenzyleyanide) and C.A.P. (chloroacetophenone). They cause immediate irritation of the eyes, profuse lacrimation and blepharospasm. The conjunctiva is injected and swollen, but there is usually no involvement of the cornea. The symptoms generally disappear in a few hours, and the eyes respond well to lavage with bland lotions. Chlorine, phosgene and other chemical compounds also cause conjunctival irritation.

Mustard gas (dichlorethyl sulphide) usually produces ocular symptoms after a latent period of from 6 to 8 hours, and is effective when very dilute (1 in 10,000,000 in air). In moderately severe cases the conjunctiva is congested and swollen in the interpalpebral area. Functional blepharospasm may persist after all inflammatory signs have disappeared, and fear of blindness may delay convalescence. In more severe cases the interpalpebral zone of conjunctiva is white from coagulated exudate, and chemotic conjunctiva bulges forwards from the fornices. The lids are swollen and stuck together by discharge. The cornea is stippled ("orange-skin" cornea), with cedematous and roughened epithelium, and corneal sensation is diminished; or the interpalpebral strip may necrose. Many cases break down with recurrent corneal ulceration fifteen to twenty years later (*delayed mustard gas keratitis*).

Arsenical vesicants are much more destructive in their action. They cause rapid necrosis of the conjunctival and corneal tissues with which they come into contact resulting in dense corneal opacities or even perforation of the eye.

The *treatment* of conjunctivitis caused by lacrimatory gases is irrigation with bland lotions—normal saline or sodium bicarbonate (2 per cent.). The eyes should not be bandaged, but dark glasses used.

In mustard gas injuries the lids should be gently separated, the cornea inspected and the patient assured that he is not blind. The eyes are irrigated with warm sodium bicarbonate lotion and emollient drops instilled; dark glasses are worn, and a bland ointment is applied to the lid margins at night. If the cornea is hazy or stains with fluorescein, atropine (1 per cent.) should be instilled twice daily with the same precautions as described for hypopyon ulcer (p. 198). Antibiotics may be given if there is any evidence of infection. Tarsorrhaphy may be necessary in some cases. It is very important to combat the anxiety by appropriate means. At a later stage contact lenses afford protection to the cornea and aid vision. In delayed mustard gas keratitis, lamellar keratoplasty gives the best results.

Arsenical vesicants are neutralized by the local application of BAL ointment (British Anti-lewisite) which, however, is only effective if it is applied within a few minutes of the injury.

CONTUSIONS

Injuries by blunt instruments vary in severity from a simple corneal abrasion to rupture of the globe. A great number of lesions may result; indeed, every part of the eye may be so injured by a contusion as seriously to diminish vision. Moreover, in some cases the changes are delayed or progressive so that *in all cases a guarded prognosis should be given*.

As a general rule either the anterior segment of the eye in front of the iris-lens diaphragm, or the posterior half, is preferentially affected. The mechanism is as follows. When a force impinges upon the cornea this tissue is thrust inwards and may even be forced against the lens and iris; the wave of aqueous pushes these

structures backwards and as the compression wave rebounds from the back of the eye, they are thrust forwards again. In this way they may be severely traumatized ; and at the same time the wave of pressure, striking the retina and choroid, may do considerable damage. Delayed complications such as secondary glaucoma, hæmophthalmitis or traumatic iridocyclitis may follow.

The various conditions resulting therefrom will now be briefly enumerated.

The cornea may suffer an abrasion, deep opacities may develop, or partial or complete rupture may occur.

A simple *abrasion* may be caused. It is recognized by distortion of the corneal reflex and by the use of fluorescein (p. 101). There is much pain similar to that due to the presence of a foreign body, increased on moving the lids, much lacrimation and reflex blepharospasm. It may become infected and give rise to a corneal ulcer, especially if a mucocele is present (p. 511). In the simple cases the use of a bland lotion and ointment to prevent the lids sticking together, and a pad and bandage for a few days, suffice. Ulceration must be suitably treated (p. 192).

Recurrent Erosion (Recurrent Traumatic Keratalgia) may occur spontaneously but is particularly liable to happen after scratches especially with babies' finger-nails ; in many cases, however, it is indicative of a degenerative condition of the cornea. The abrasion, however produced, usually heals quickly, but is followed some days, weeks, or even months later, by acute pain and lacrimation, generally on first opening the eyes in the morning. If the cornea is then stained with fluorescein an abrasion will be found, usually at the original site but sometimes elsewhere, or there may be one or a group of vesicles. The attack rapidly passes off with appropriate treatment, but often recurs again and again, particularly on awakening in the morning. There is no doubt that in these cases the epithelium is abnormally loosely attached to Bowman's membrane, and is liable to be torn off by the lid on waking. Early attacks should be treated in the same manner as a simple abrasion, but if the attacks are repeated the spots should be curetted and touched with a strong alcoholic solution of iodine or pure carbolic acid (p. 193) ; if this fails the mild application of X-rays has been found beneficial, and in the worst cases recourse may be had to lamellar keratoplasty.

A *deep opacity* in the substance of the cornea may result from a contusion, usually in the form of delicate grey striæ interlacing in different directions, due to œdema of the corneal stroma or occasionally to wrinkling of Descemet's membrane (p. 221). It generally clears up without leaving a permanent opacity. There may be ruptures in Descemet's membrane followed by acute œdema of the stroma ; owing to its elasticity the edges are rolled over and the deposition of fine granules of uveal pigment on the posterior surface of the cornea is common.

Blood-staining of the cornea occasionally results from a contusion which has caused hæmorrhage into the anterior chamber usually accompanied

by a rise of tension of the eye. The whole cornea is at first stained, the colour varying according to the duration of the condition. It may be reddish-brown, or greenish ; in the latter case the condition simulates dislocation of the clear lens into the anterior chamber (p. 380). The cornea gradually and very slowly clears from the periphery towards the centre, the whole process taking two years or more. Microscopically there are myriads of minute, highly refractile rods packed in the lamellæ of the stroma, and sometimes round granules of pigment in the corneal corpuscles. These are derivatives of hæmoglobin which may or may not contain iron and are removed by phagocytic action—a slow process. In the absence of other causes of defective vision, sight may eventually be completely restored but is usually permanently impaired.

Rupture of the cornea is very rare ; an attempt may be made to save the eye by suturing the cornea with an atraumatic needle.

Sclera. *Rupture of the globe* is generally due to its being suddenly and violently forced against the orbital walls. It is often caused by a fall upon some projecting object, such as a knob or a key in a door, and in country districts by a blow from a cow's horn. The force usually comes from the direction down and out, where the eyeball is least protected by the orbital margin ; the sclera gives way up and in at its weakest part, in the neighbourhood of the canal of Schlemm. The wound runs obliquely from the canal outwards and backwards through the sclera to appear more or less concentric with the corneal margin and about 3 mm. behind it. The conjunctiva is often intact, but there are always severe injuries to other parts of the eye. The iris is generally prolapsed or torn away (iridodialysis) or retroflexed (*vide infra*). The lens may be expelled from the eye or escape under the conjunctiva (subconjunctival dislocation of the lens) or be forced back into the vitreous, in which case the anterior chamber becomes deep. Intra-ocular bleeding may be profuse, filling the anterior chamber and vitreous, and the condition may be complicated by a detachment of the retina with or without subretinal or subchoroidal hæmorrhage. Ultimately, the eye usually shrinks and is lost.

Treatment. The eye must be carefully examined, using lid retractors, under an anæsthetic if necessary. In severe cases nothing remains but to excise the collapsed globe. In less severe cases without extrusion of the intra-ocular contents, the wound should be sutured, atropine instilled and the patient kept in bed. If the rupture involves the periphery of the cornea the iris alone may be prolapsed. It is then a good plan to insert the sutures in the sclera without tying them, before excising the prolapse ; thereafter they are tied.

In subconjunctival dislocation of the lens it seems tempting to open the conjunctiva and let out the lens. This is, however, contra-indicated in the early stage ; such a procedure will almost inevitably

involve the escape of vitreous and possibly panophthalmitis. The lens will gradually become absorbed, but no harm will accrue if the remnants are removed after the scleral rupture has healed.

The iris and ciliary body may suffer functional defects or may be actually torn.

A *traumatic miosis* due to irritation of the nerves occurs initially in every severe contusion; sometimes the condition persists for some time, frequently accompanied by a spasm of accommodation and a consequent myopia. This may be followed by a traumatic mydriasis often associated with paralysis of accommodation.

In *traumatic mydriasis* following a contusion, the pupil is large and immobile and usually remains moderately dilated permanently. It is probably due to paralysis of the motor nerve fibres which may be stretched or torn in their passage through the ciliary body.

The substance of the iris is often torn. The most common lesions are minute *ruptures in the pupillary margin* which are of little

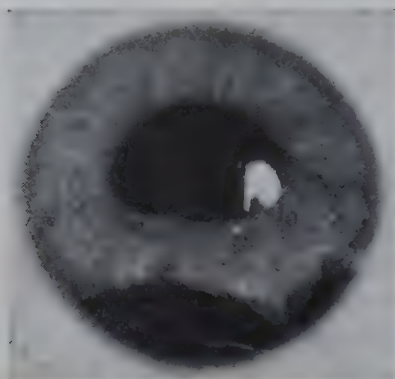


FIG. 277. Iridodialysis.

significance; *radiating lacerations* of the iris, sometimes extending to the ciliary margin, are rare. *Iridodialysis*, wherein the iris is torn away from its ciliary attachment for a variable distance, occurs more frequently (Fig. 277). A black biconvex area is seen at the periphery, and the pupillary edge bulges slightly inwards. With the ophthalmoscopic mirror a reflex can be obtained through the peripheral gap, and the fibres of the suspensory ligament and the edge of the lens may be visible. Uniocular diplopia may be produced by this injury. In extensive iridodialysis the detached portion of the iris may be completely rotated so that the pigmented back of the iris faces forwards (*anti-flexion of the iris*). The iris becomes re-attached only in exceptional cases, but, apart from other injury, the lesion rarely causes serious results. In *traumatic aniridia* or *irideremia* the iris is completely torn away from its ciliary attachment, contracts into a minute ball, and sinks to the bottom of the anterior chamber, where it may be invisible. Rarely the same appearance is caused by *total inversion* or *retroflexion* of the iris, the whole iris being doubled back into the ciliary region out of sight. More commonly inversion is

partial so that the appearance of a coloboma (*q.v.*) is obtained, but the fibres of the suspensory ligament cannot be seen. The ciliary body may also be torn or ruptured near its anterior attachment resulting in its subsequent retraction with a tendency to glaucoma. In all these cases there is usually a hyphæma and other injuries may be present.

The *treatment* consists of rest. Atropine should be instilled in iridodialysis, but not in ruptures of the iris or if the lens is subluxated. If the iridodialysis is gross and causes symptoms such as diplopia, the torn peripheral edge of the iris may be anchored with a silk suture into a scleral incision just behind the limbus.

The lens may show cataractous changes or be dislocated.

In some cases a circular ring of faint or stippled opacity is seen on the anterior surface of the lens due to multitudes of brown amorphous granules of pigment lying on the capsule (*Vossius's ring*). It usually has about the same diameter as the contracted pupil, and is due to the impress of the iris on the lens, produced by the force of the blow driving the cornea and iris backwards. Minute discrete subcapsular opacities may be seen after resorption of the pigment.

Concussion Cataract may assume many varied forms. It is due partly to the mechanical effects of the injury on the lens fibres and largely to the entrance of aqueous due to damage to the capsule, either impairment of its semi-permeability or often the result of actual tears which, particularly if they are small and peripheral, may not be clinically visible. They frequently occur at the thin portion of the capsule covering the posterior pole of the lens. Sometimes, especially if they are covered by the iris, such tears are rapidly sealed, at first with fibrin and later by the proliferation of the subcapsular epithelium which secretes a new capsule; in these cases the entrance of aqueous is stopped and the opacity in the lens may remain stationary or even regress. Alternatively, the tear may remain open and opacification progress to involve the entire lens.

The most common manifestation of concussion cataract is the appearance of a number of discrete *punctate opacities* in the superficial layers of the cortex. These may be segmental in distribution and usually remain stationary; but in old people, or as age advances, the changes characteristic of senile cataract may slowly evolve, sometimes after the lapse of some years.

The most typical appearance is that of a *rosette-shaped cataract*, usually in the posterior cortex, sometimes in the anterior or both (Figs. 278 and 279). In this condition an accumulation of fluid marks out the architectural arrangement of the lens (p. 9). The star-shaped cortical sutures are therefore delineated and from them radiate feathery lines of opacities outlining the lens fibres. Occasionally the rosette disappears; sometimes it remains stationary and sometimes it progresses to total opacification of the lens. a

complication which may appear rapidly within a few hours after the injury or may be delayed for many months.

A *late rosette-shaped cataract* may develop in the posterior cortex one or two years after a concussion. It is smaller and more compact than the early type and its sutural extensions are short (Fig. 280).

The treatment of such cataracts is on general lines (p. 275) but, unless the rapid intumescence of the lens leads to a secondary glaucoma which may then require immediate measures for its relief (p. 296), any surgical interference should be delayed for some months until the final outcome is apparent. If possible, the eye should be left until all signs of inflammation have subsided, whereafter it should be treated as indicated for unilateral cataract (p. 278).

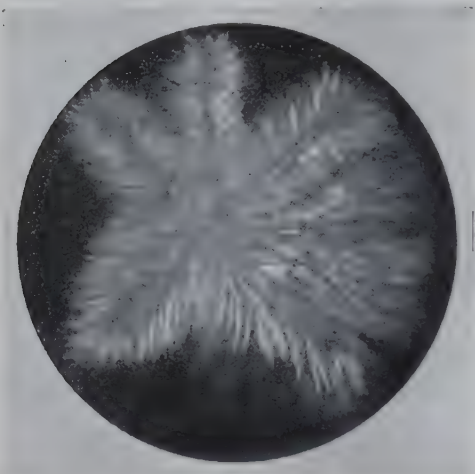


FIG. 278. A concussion rosette (D. Cattaneo).



FIG. 279.

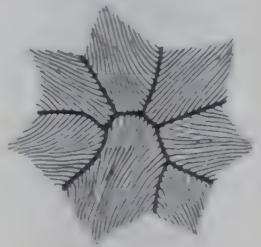


FIG. 280.

FIG. 279. The arrangement of the feathery opacities in an early concussion rosette. The leaves of the rosette are formed by the suture acting as a backbone from which the opacities radiate outwards. FIG. 280. The arrangement in a late rosette. The feathery opacities stream outwards from the sutures which lie in the angles between neighbouring leaves.

Dislocation of the lens may occur when the relatively fragile suspensory ligament is torn by the to-and-fro wave of pressure set up by the contusion. If the tear is partial the lens may be *subluxated* so that it is displaced laterally and sometimes slightly rotated. This leads to a variation in the depth of the anterior chamber which is deeper in the part unsupported by the lens. With the pupil dilated, the edge of the lens may be seen as a grey convex line by oblique illumination, but more readily and unmistakably as a black line with the ophthalmoscope (p. 119). The lack of support to the iris causes tremulousness when the eye is moved.

If the rupture to the suspensory ligament is complete the lens is *dislocated*, usually into the vitreous. Sometimes it remains clear and can be seen only with difficulty; at other times it turns opaque and appears as a yellow mass. Alternatively, particularly if the blow has been slanting, the lens is dislocated into the anterior chamber, an accident which may follow a trivial injury if the lens

is shrunken. A clear lens in the anterior chamber is not always easily recognized, but it does not long remain clear and diagnosis is then easy. It is more globular than normal owing to its freedom from the restraint of the suspensory ligament. When still clear it looks like a globule of oil in the anterior chamber. With oblique illumination it has a golden rim, due to total reflection of the light ; this is the exact opposite of the total reflection when the edge of the lens is seen with the ophthalmoscope, the light being then totally reflected away from the observer's eye. The lens in the anterior chamber causes spasm of the sphincter pupillæ, which may occur at the moment when it is passing through the pupil. An iridocyclitis or an intractable secondary glaucoma is then set up so that the eye is usually lost if the anteriorly dislocated lens is allowed to remain.

Dislocation of the lens always causes a considerable disturbance of vision. In subluxation there is astigmatism which is much increased by tilting of the lens. The slackening of the suspensory ligament causes increased curvature and myopia which, however, may be more than compensated by backward displacement.

If the lens is displaced so much laterally that the edge crosses the pupil, uniocular diplopia is present. Through the aphakic area of the pupil the eye is highly hypermetropic, through the phakic portion it may be myopic, in addition to which the periphery of the lens acts as a prism. Ophthalmoscopic examination by the indirect method in these conditions shows two images of the disc differing considerably in size, and by the direct method the fundus may be observed through the phakic or through the aphakic portion of the pupil. In total dislocation into the vitreous the effect is that of the old cataract operation of couching ; the pupillary area is aphakic and the refraction is highly hypermetropic, requiring cataract spectacles for its correction. In these cases the vision may be retained for many years.

Treatment. In dislocation forwards miotics must be avoided, since the contraction of the iris behind the lens may actually induce secondary glaucoma. In the absence of irritation vision may be improved by suitable glasses in cases of total luxation into the vitreous and subluxation. In the latter case it is usually impossible to correct the astigmatism so that a correction for the aphakic part of the pupil may give better visual results. If iridocyclitis or secondary glaucoma develops the lens should be extracted if it is possible ; this is imperative when the lens is in the anterior chamber but in all cases it is unusually difficult. There is always a considerable rupture of the suspensory ligament, so that vitreous presents as soon as the corneo-scleral section is completed, and the delivery of the lens has usually to be effected with the scoop, often involving the loss of some vitreous (p. 432). If extraction is impossible, an iridectomy or trephining may improve matters, but more usually fails. If the eye is blind and painful it should be excised.

The vitreous is usually disorganized to some extent. The

most common occurrence is the appearance of clouds of fine *pigmentary opacities*, the vitreous frame-work when examined with the slit-lamp being bespangled with innumerable golden-brown dots derived from the uvea. In severe concussions complete liquefaction of the gel is not uncommon.

Hæmorrhage into the vitreous is also common. The whole vitreous chamber may be filled with blood so that no reflex will be obtained with the ophthalmoscope, but with oblique illumination a dull red hue may be seen, especially if the pupil is dilated. The blood may become almost completely absorbed but cloudy opacities usually remain. In rare cases proliferative retinopathy follows from organization of the clot (p. 312).

The choroid may be torn or hæmorrhages may occur.

Rupture of the choroid follows a severe contusion by a blunt body striking the front of the eye ; a similar result may follow the concussion effect of a bullet passing through the orbit behind the eye. Immediately after the injury the view is obscured by extravasation of blood. When it has become absorbed the rupture, usually not far from the disc, concentric with it and on its temporal side, is seen as a curved white streak over which the retinal vessels pass and which rapidly becomes pigmented along its edge (Plate XIX, Fig. 2) ; the white appearance is due to the sclera shining through. Sometimes multiple ruptures occur more or less concentric with each other. If the choroid is ruptured near the macula, loss of central vision results, but simple ruptures of the choroid in which the macula is not involved cause little impairment of vision. The treatment consists of the instillation of atropine and rest until the extravasated blood is absorbed.

A contusion may cause a *choroidal hæmorrhage* ; this may either be small, marked later by patches of choroido-retinal atrophy, or large, either subretinal or subchoroidal. The latter can seldom be seen ophthalmoscopically and is usually associated with more extensive damage.

The retina may suffer œdematous changes, be torn, or degenerative changes or hæmorrhages may occur.

Commotio retinæ (*Berlin's œdema*) is a common result of a blow on the eye. A milky-white cloudiness due to œdema appears over a considerable area at the posterior pole (Plate XIX, Fig. 1). Sometimes it disappears after some days when vision is usually restored. In other cases, although vision may be good at first, central vision gradually diminishes, the loss of function being associated with the development of pigmentary deposits at the macula.

Traumatic macular degeneration may often appear slight and the fine pigmentary changes are easily overlooked immediately after the accident. The pigmentation, however, which mainly aggregates at the fovea, has a tendency to increase progressively and has a serious and permanent effect on central vision. Alternatively, the

PLATE XIX
TRAUMATIC LESIONS OF THE FUNDUS

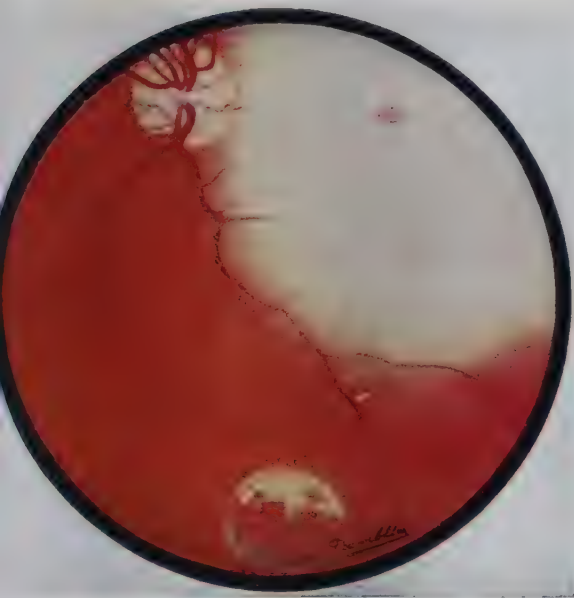


FIG. 1. Berlin's œdema with an area below of traumatic chorioretinopathy. (Doherty.)

FIG. 2. Ruptures in the choroid.



FIG. 3. Traumatic chorioretinopathy after a bullet wound of the orbit.

PLATE XX
INTRA-OCULAR FOREIGN BODIES

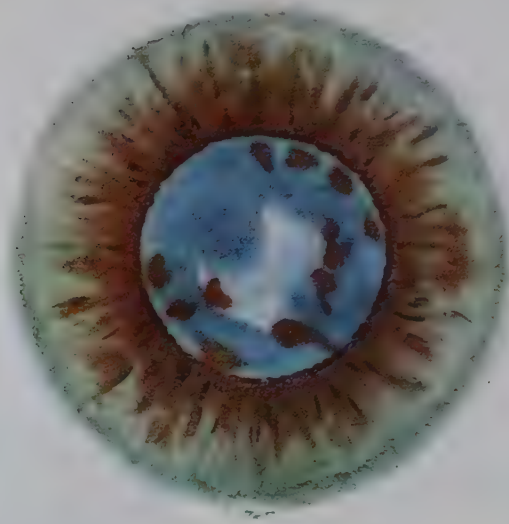


FIG. 1. Siderosis.

Note the rust-staining of the iris, the patches of rusty deposit on the lens capsule and the cataract.

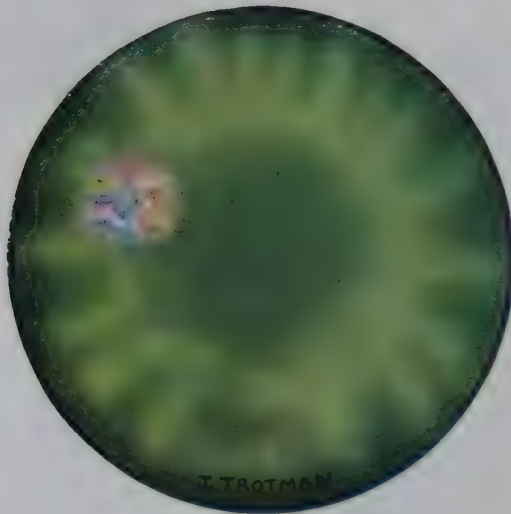


FIG. 2. Chalcosis. Sun-flower cataract.

œdematous changes may lead to the formation of cystic changes at the macula, and on the rupture of a cyst a *macular hole* may be formed. Either appears clinically as a round or oval, deeply red patch just as if a hole has been punched out. The differentiation is not easily made unless focal examination of the fundus is made by the slit-lamp (Plate IV, Figs. 3 and 4). In cystic formation some central vision may remain ; in the case of a hole central vision is lost.

Tears in the retina may follow a contusion, particularly in the peripheral region in eyes already suffering from myopic or senile degeneration. It has already been pointed out that these are frequently the precursors of *retinal detachments* (*q.v.*). Sometimes such ruptures are very large, particularly when the retina is torn at the periphery to form an anterior dialysis.

Occasionally, particularly in the concussion injuries associated with gunshot wounds, a rupture of the retina is associated with a similar rupture of the choroid. Such cases present a characteristic picture of *traumatic proliferating chorioretinopathy* (Plate XIX, Fig. 3). There is much hæmorrhage, usually into the vitreous, and the mesodermal tissues in the choroid provide abundant material for fibrous organization. Contrary to the usual result of retinal hæmorrhages into the vitreous, therefore, there is dense proliferation of fibrous tissue which projects in a great mass into the vitreous and may extend as thick bands over large areas of the retina. Grave visual disability usually results.

Partly because of the progressive macular changes, and partly because of the possibility of the formation of small peripheral retinal tears which may lead subsequently to a retinal detachment, the prognosis should be guarded in *all* cases of serious blows upon the eye.

Optic nerve. The optic nerve is not infrequently injured in fractures of the base of the skull (p. 532). Injuries by sticks, knives or other penetrating instruments are rare. Avulsion of the optic nerve is very rare in civilian life but occurs in gunshot wounds of the orbit (p. 532).

The tension may be seriously disturbed following concussion injuries, particularly if they are severe. A condition of *hypotony* or, alternatively, of *traumatic glaucoma* may supervene. Both of these may result from the profound vasomotor reaction which follows a contusion—an initial vasoconstriction followed by an intense vasodilatation ; other causes may be operative (dislocated lens, hæmorrhage, etc.). Such a glaucoma may be controlled by miotics, but sometimes, particularly when complicated by hæmorrhages, it is intractable even to operative treatment so that the permanent visual loss may be profound or complete. Since it is frequently associated with other gross ocular damage, eventual excision of the globe is often advisable.

A violent and intractable *post-traumatic iridocyclitis* is not un-

common ; sometimes it is due to the irritation caused by the end-products of intra-ocular hæmorrhages (*hæmophthalmitis*). In either case it is often unusually resistant to treatment, although in the early stages, in addition to the usual methods, cortisone preparations (drops, ointment or subconjunctival injections) (p. 145), frequently have a dramatic effect. If the condition becomes thoroughly established, however, it may terminate in atrophy and phthisis bulbi.

PERFORATING INJURIES

Perforating injuries caused by sharp instruments or foreign bodies are all potentially serious and should be treated as emergencies. Their seriousness arises from four causes.

(1) The *immediate trauma*. This will be considered in the following sections.

(2) The introduction of *infection*. Occasionally in a corneal wound caused by a dirty implement, pyogenic organisms are carried into the eye, multiply there and cause rapid necrosis of the whole cornea. In these cases a ring of deep infiltration appears 2 or 3 mm. internal to and concentric with the corneo-scleral margin—so-called *ring abscess*. If the organism is the *Ps. pyocyanea*, there is much chemosis of the conjunctiva sometimes with a greenish discharge. Usually panophthalmitis is set up and the whole of the central part of the cornea is cast off. The only chance of saving such an eye is by a paracentesis directly the infiltration is observed and the institution of intensive treatment with polymyxin or Soframycin (p. 144). In other infections the appropriate antibiotic should be used (*e.g.*, penicillin introduced into the anterior chamber, followed by subconjunctival injections).

If it finds access into the anterior chamber, pyogenic infection leads to a purulent iridocyclitis with hypopyon (p. 195) and usually panophthalmitis (p. 243). The treatment of these conditions has already been indicated and it will be remembered that even although the infection itself may be controlled by suitable antibiotics, it is rare to save a useful eye once suppuration has become thoroughly established. This applies more particularly to infections of the vitreous, and in these circumstances if the lens is wounded the chances of saving the eye are greatly diminished. In view of this it is a good rule to anticipate the onset of infection in every case of perforating injury when contamination may be suspected by prophylactic antibiotic treatment. At this stage a good combination is a systemic course of one or other of the sulphonamides combined with subconjunctival injections of penicillin or Soframycin.

Two special infections should be noted. *Gas-forming organisms*, such as *Cl. welchii*, are occasional contaminants. They excite a virulent panophthalmitis with a brownish discharge and gas bubbles in the anterior chamber. Although they are sensitive to penicillin

destruction of vision has always followed. *Tetanus* is a rare complication ; if this infection is introduced into the eye, the localized cephalic type of tetanus often results. Whenever such a complication may be suspected, as in agricultural or road accidents, if the patient is not already immunized, prophylactic treatment should be instituted.

(3) *Post-traumatic iridocyclitis* is a common sequel to a perforating wound. There is always a reactive inflammation at the time of injury which often clears ; but frequently this is followed by a recalcitrant plastic iridocyclitis which tends to progress steadily—often with intermissions and relapses—sometimes for years, resulting not uncommonly in atrophy and phthisis bulbi. The presence of much hæmorrhage intensifies the tendency (*hæmophthalmitis*). In both the immediate and delayed type of post-traumatic iridocyclitis, treatment by cortisone preparations locally (p. 145), in addition to the usual methods (atropine, heat, etc.), frequently has a dramatic, although often temporary effect.

(4) *Sympathetic ophthalmitis*, one of the most dreaded complications of perforating wounds, will be discussed subsequently (p. 400).

A wound with a sharp instrument may penetrate the cornea, the corneo-scleral junction or the sclera : it may pass in for a variable distance, wounding the iris or lens, or pass through the eye.

Wounds of the conjunctiva are common. They heal readily, but the process may be hastened and the resulting adhesion to the sclera lessened by introducing one or more sutures. Polypoid masses of granulation tissue sometimes form on the surface ; they should be snipped off with scissors after the application of Pantocaine.

Wounds of the cornea may be linear or lacerated. The margins swell up soon after the accident and become cloudy through imbibition of fluid thus facilitating closure of the wound and restoration of the anterior chamber. If small and limited to the centre, corneal wounds heal well unless they become infected ; the eye is irrigated with sterile saline, atropine instilled, an antibiotic ointment inserted, and a pad and bandage applied. A permanent dense opacity is left, and the contraction of the organizing scar tissue causes irregular astigmatism. If the wound becomes infected it must be treated like a perforating ulcer.

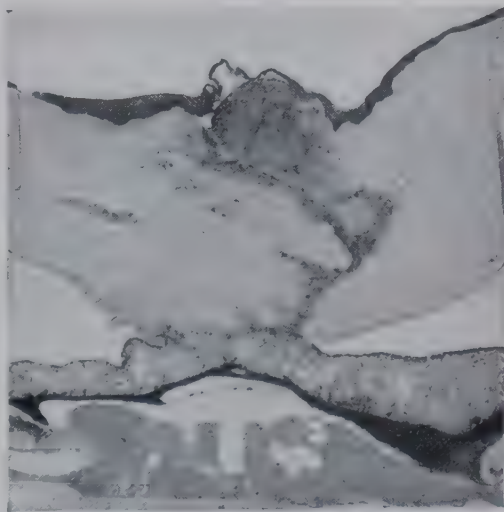


FIG. 281. Wound in the cornea showing an adhesion of the iris to its under surface (Ashton).

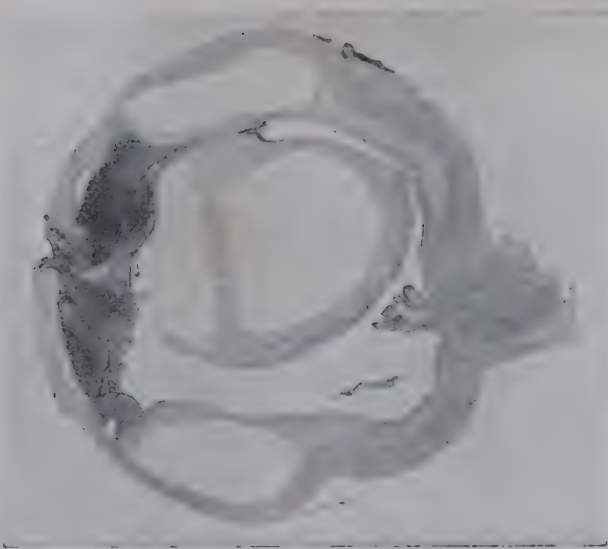


FIG. 282. A large corneal wound. Most of the uveal tissue with a mass of hæmorrhage has prolapsed and the interior of the eye is disorganized (Ashton).

If the wound is large an adhesion of the iris or its prolapse is almost certain to occur (Figs. 281-2). The prolapsed iris should never be replaced, even if this is possible, since it may carry infection into the eye. It must be abscised (p. 195); the technique of this operation will be discussed subsequently (p. 415). In large wounds, which are often corneo-scleral in type, healing may be facilitated by covering with a conjunctival flap or, more effectively, by closure with

sutures. In the latter case sharp atraumatic corneo-scleral needles should be used, the sutures should never penetrate beyond two thirds of the corneal thickness lest epithelial ingrowth occur, and they should not be tied too taut lest the enclosed tissue necrose.

A suitable conjunctival flap depends on the site of the wound. If it is central a pedicle flap may be employed.

If the wound is peripheral, in which case it often involves the sclera as well, a limbal flap may suffice (Figs. 283-5). Before abscission of the prolapse of the iris which is almost always present, the conjunctiva is freed at the limbus and undermined (Fig. 284) and, after the prolapse has been dealt with, drawn over the wound and secured by stitches as in Fig. 285. If the wound is mainly scleral, it should be freely exposed as in Fig. 286. The edges of the scleral wound are carbolyzed and sutures inserted by an eyeless corneo-scleral needle and looped ready to tie. Any prolapsed iris and ciliary body are drawn out of the wound and abscised and the scleral sutures are immediately tied so as to minimize loss of vitreous. Finally, the wound is covered with conjunctiva. All these operations should be done under regional analgesia and full akinesia (p. 407). Afterwards atropine and antibiotics are instilled, the eye bandaged lightly, and the patient put to bed.

If in a few days it is found that there is an anterior synechia, this should usually be divided as soon as the wound is sufficiently healed to permit the necessary procedures without re-opening it. If this is not done, the traction on the iris may keep up irritation in the eye and increase the liability to sympathetic ophthalmitis. Moreover, a broad anterior synechia predisposes to secondary glaucoma, secondary infection and panophthalmitis.

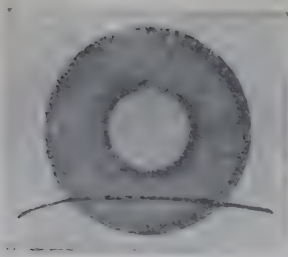


FIG. 283.

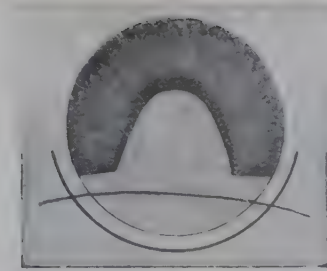


FIG. 284.

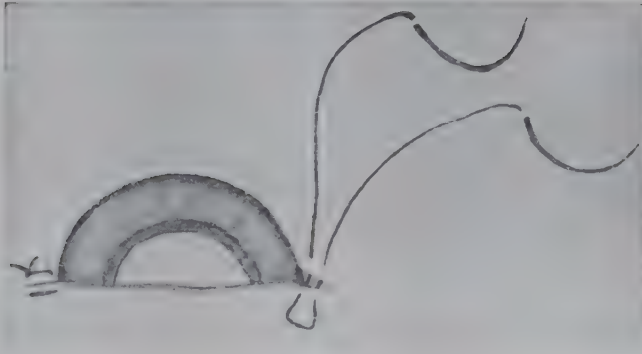


FIG. 285.

FIGS. 283-5. Fig. 283 illustrates diagrammatically a large scleral wound involving the cornea and sclera at either side. Fig. 284 shows an abscission of a presumptive prolapse of the iris; the curved line concentric with the lower part of the limbus indicates the incision for a conjunctival flap which is undermined, brought up over the cornea, and held in position by sutures, as in Fig. 285.

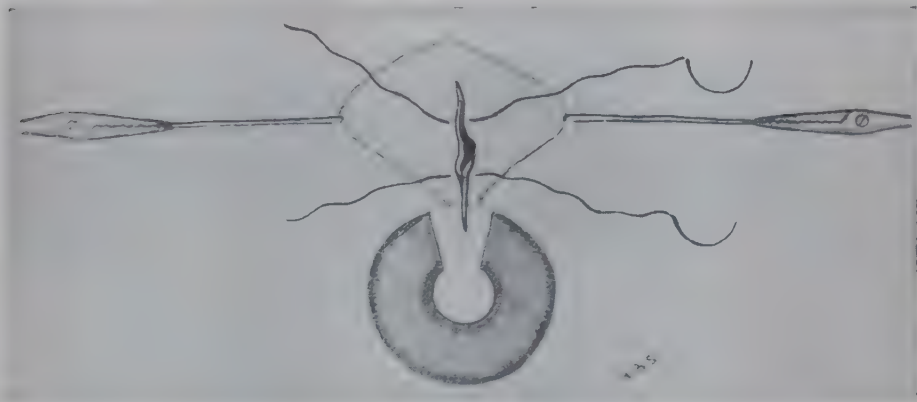


FIG. 286.

Scleral wounds some distance from the cornea are not always easily recognized. The eye may have been wounded through the lid, the bruising and laceration of which may make examination difficult. The lid should be raised from the globe and drawn back with a retractor (Fig. 95), under local analgesia. Even when

the eye is examined the effusion of blood under the conjunctiva may render the diagnosis uncertain, still more the question of perforation. When perforation has occurred, there is a reduction in the ocular tension. If the perforation is near the cornea, the anterior chamber is shallow or obliterated. If the wound is large, prolapse of some of the contents of the globe occurs; the uveal tissues—iris, ciliary body, or choroid—are most easily recognized on account of their pigmentation. Very often the gelatinous vitreous can be seen hanging out of the wound. Hyphæma and vitreous hæmorrhage may be present with or without perforation.

Treatment. If the injury is so severe that there is no likelihood of the recovery of useful vision, the eye should be excised. If there is a chance of useful vision the sclera may be sutured and the sutures tied after abscission of the prolapsed tissue. Very small wounds do not require suturing, but the conjunctiva should be cleansed and stitched over them. Atropine and antibiotics should be instilled, and both eyes bandaged. Complete rest in bed is imperative. Seriously injured eyes usually shrink unless, indeed, panophthalmitis ensues.

Wounds of the lens cause traumatic cataract and are always a serious complication. They may escape notice at first, especially if the wound is small, as that caused by a needle or thorn. If the wound in the capsule is small, the entry of aqueous causes a localized cloudiness in its vicinity and, irrespective of the site of the wound, opacities in the form of feathery lines appear in the posterior cortex, which later develop into a rosette-shaped cataract resembling that of early concussion cataract (Fig. 287). Occasionally the wound in the capsule becomes sealed, particularly if a posterior synechia



FIG. 287. Rosette-shaped cataract at the posterior pole following perforation of the lens by a foreign body. A disturbance in the lens along the track of the foreign body is seen.

develops, in which case these changes may be stationary ; more usually they are progressive until a complete cataract is formed. If this is associated with much swelling and turgescence of the lens, an occurrence common in children, secondary glaucoma may develop.

If the wound is large, events may be more turbulent. The lens rapidly opacifies and flocculent grey masses protrude through the opening in the capsule ; sometimes the whole chamber is full of white flocculi, and the entire lens nucleus may escape. The lens substance tends to be gradually dissolved by the aqueous, and in young patients the whole lens, with the exception of the capsule, may thus disappear as occurs in the operation of discission, but as a rule the process is stopped through closure of the capsular wound and the enclosed lens fibres remain opaque. In adults over thirty or thirty-five years of age the nucleus does not dissolve in this way.

A traumatic cataract of this type is liable to set up serious complications. Traumatic iridocyclitis is invariable and may be severe. The swelling of the lens, greater and more rapid the younger the patient, keeps the iris in contact with the cornea, so that re-formation of the anterior chamber is much delayed ; the abscission of any prolapsed iris in such a manner as to free it completely from the wound is difficult. The subsequent prolonged contact of the iris with the cornea facilitates the formation of broad adhesions of this tissue and often also of the lens capsule which it may be found impossible to divide, or which, if divided, quickly re-form. The presence of synechiæ and the plasmoid nature of the aqueous excite a secondary glaucoma (*q.v.*) ; while the liability to the development of infection or subsequent sympathetic ophthalmitis is much increased.

The *treatment* of traumatic cataract in the absence of secondary glaucoma is by rest in bed, atropine, and a bandage. It is of the utmost importance that the pupil should be kept well dilated to prevent the formation of synechiæ. *Sterile* atropine ointment, 1 per cent., should be used three or four times daily, and if this is not effective every expedient should be tried to maintain mydriasis—the insertion of a crystal of atropine into the conjunctival sac, the subconjunctival injection of mydraine (p. 563), four-hourly hot bathings, or medical diathermy. Topical cortisone preparations (p. 145) may control the inflammatory reaction.

As a rule, except sometimes in the case of children, operative measures are necessary to deal with the opaque lens, but these should be delayed until the eye is quiet and white, if delay is possible. Even in children discission is often necessary to cause complete absorption ; in adults with a hard nucleus, extraction may be required (p. 425). In either case a subsequent needling is usually necessary in order to obtain an opening in the capsule through which vision is made possible. If raised tension does develop, the swollen lens substance must be got rid of by a curette evacuation (p. 425).

In all cases of perforating injury the closest watch must be kept upon the wounded eye and its fellow lest subsequent complications develop, especially sympathetic ophthalmitis. If there is much prolapse of vitreous as well as of iris and ciliary body, especially if the lens is also wounded, there is little probability of saving the vision (Fig. 282). If it is almost certain that useful vision will be lost, the risks of sympathetic inflammation should not be incurred and the eye should be promptly excised.

If, despite treatment, the eye does not show signs of quietening in the course of a week or ten days, as shown by the diminution of ciliary injection and the cessation of photophobia and lacrimation, it should be excised. In the interval the cornea is examined most carefully each day with the slit-lamp for precipitates ("k.p."); if they are seen the eye should be excised. Similar care is devoted to the discovery of spots of "k.p." in the other eye, and if they should be found there, intensive topical steroid treatment should be immediately started; in this event excision of the injured eye is still more imperative at the earliest possible moment. If the eye quiets down quickly and there is no evidence of iridocyclitis in either eye, the case will probably do well, but it should be kept under observation for many months.

PERFORATING WOUNDS WITH THE RETENTION OF FOREIGN BODIES

The retention of a foreign body adds considerably to the danger and anxieties of a perforating injury. The foreign bodies most likely to penetrate the eye and be retained are minute chips of iron or steel (accounting for 90 per cent. of the foreign bodies in industry), stone, and particles of glass, lead pellets, copper percussion caps, less frequently spicules of wood. In chipping stone with an iron chisel, it is usually a chip of the chisel and not of the stone which enters the eye. Among war injuries penetration of the eye by fragments of the casing of rifle bullets, often containing nickel, of shells, bombs and other missiles frequently occurs.

The *size and velocity* of the missile are of importance. If the foreign body is large so much damage is usually caused that the eye has to be removed. Very minute particles can, however, penetrate the cornea or sclera and lodge in the deeper parts of the eye. The velocity of these small particles must be very high, for the energy needed to penetrate the walls of the globe is considerable; it will be remembered that the relationship of energy to mass and velocity is given by the formula, $E = mv^2/2g$, so that when the mass is small, the penetrating power depends on speed.

The entrance of a foreign body into the eye may cause damage in one of three ways—by mechanical effects, by the introduction of infection, and sometimes by specific action (chemical and otherwise) on the intra-ocular tissues. We will first consider the mechanical effects.

The foreign body may enter the eye either through the cornea or sclera. Having penetrated the cornea it may be retained in the anterior chamber where it may fall to the bottom and, if very small, lie deeply in the angle hidden by the scleral ledge where it can only be seen gonioscopically. It is generally, however, caught in the iris, and can be recognized with a loupe. A piece of glass in the anterior chamber is exceptionally difficult to see because its refractive index differs so little from that of the surrounding media ; for its recognition the slit-lamp is usually necessary.

The foreign body may pass into or through the lens, either by way of the iris or of the pupil. In each case a traumatic cataract is produced, which undergoes the usual changes (p. 388). If the particle has passed through the iris there will be a hole in this structure ; this looks black by oblique illumination, but shows a red reflex when illuminated by the ophthalmoscope, unless the lens behind the hole is cataractous. A hole in the iris is of great diagnostic significance, since it rarely occurs except as the result of perforation by a foreign body. The foreign body may be visible in the lens, either before or after dilatation of the pupil, but it is possible for it to pass through the iris and through the circumlental space without wounding the lens.

The foreign body may be retained in the vitreous to which it may obtain access by various routes : through the cornea, iris and lens ; through the cornea, pupil and lens ; through the cornea, iris and zonule ; or directly through the sclera. If it comes to rest in the vitreous it may remain suspended for some time but eventually sinks to the bottom of the vitreous chamber owing to degenerative changes in the gel which lead to liquefaction, partial or complete. Sometimes air is carried in with the foreign body and appears as bubbles in the vitreous ; these rapidly become absorbed. If the particle is small, the lens clear, and there has been little hæmorrhage, the body may be seen ophthalmoscopically in the vitreous or retina ; the track through the vitreous often looks like a grey line. Most commonly the particle has enough energy to carry it directly onto the retina where it may ricochet once or even twice before it comes to rest ; occasionally it pierces the coats of the eye and comes to rest in the orbital tissues (*double perforation*).

If it lies in the retina, the foreign body, generally black and often with a metallic lustre, is surrounded by white exudate and red blood-clot, but eventually fibrous tissue usually encapsulates it and the retina in the neighbourhood becomes heavily pigmented. The degree of encapsulation depends on the amount of necrosis caused at the site of impact and is greatly assisted by the presence of mild sepsis.

The lodgement of a foreign body in the posterior segment frequently leads to degenerative changes, apart altogether from its chemical nature, which may considerably damage the sight. These may

entail a widespread degeneration, but most frequently fine pigmentary disturbances at the macula, often the result of concussion (*q.v.*), diminish or destroy central vision. The vitreous usually turns fluid, bands of fibrous tissue may traverse it along the path of the foreign body, hæmorrhage may be extensive, and retinal detachment may follow. Particles greater than 2 mm. in size usually lead to destruction of the eye (Fig. 288).

Infection. As with other perforating wounds, the introduction of infection is an ever-present danger when a foreign body enters the eye. Some types of foreign body are more likely to be associated with infection than others. Fortunately, owing to the heat generated partly on their emission and partly by their rapid transit through the air small flying metallic particles are frequently sterile,

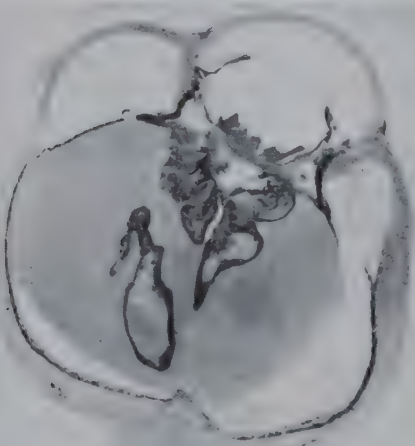


FIG. 288. The disorganization of the eye caused by the entry and retention of a copper foreign body. Purulent ophthalmitis has developed (U.S. Armed Forces Inst. of Path., Washington).

and it is notorious that infections are more likely to follow the introduction of pieces of stone or wood than anything else. Moreover, the incidence admirably exemplifies the role played in such infections by the resistance of the tissues, for the patients who develop infection are usually either old or debilitated. With regard to intra-ocular infections, it is to be borne in mind that the lens substance and the vitreous form excellent culture media, and further, that even saprophytic organisms like the *bacillus subtilis* are capable of setting up a suppurative inflammation in the eye. The occasional occurrence of gas-gangrene or tetanus infection should be remembered (p. 384). Once it has developed, the infection should be treated as in perforating wounds; despite treatment, the prognosis in terms of vision is seldom good.

The reaction of the ocular tissues to a foreign body varies with its chemical nature. Non-organized materials are either (1) inert, or (2) excite a local irritative response which leads to the formation of fibrous tissue, often resulting in encapsulation, or (3) produce a

suppurative reaction, or (4) cause specific degenerative effects. Organized material, on the other hand, tends to produce a proliferative reaction characterized by the formation of granulation tissue. Although the inert materials cause little or no reaction at the time, iridocyclitis and disorganization may eventually develop.

Glass, plastics and porcelain are inert. *Stone* may occasionally give rise to chemical changes depending on its composition. Of the metals, *gold, silver, platinum and tantalum* are inert. *Lead*, usually occurring as shot-gun pellets, becomes coated with the carbonate and excites little reaction. *Aluminium* frequently becomes powdered and excites a local reaction; so also does *zinc* which may excite suppuration—a reaction often associated with *nickel* and constantly with *mercury*. Iron and copper, the two most common materials found, undergo electrolytic dissociation and are widely deposited throughout the eye causing important degenerative changes.

Iron causes the condition of **siderosis**; so also does steel in proportion to its ferrous content. The condition is probably due to the electrolytic dissociation of the metal by the “current of rest” in the eye, which disseminates the metal throughout the tissues and enables it to combine with the cellular proteins, thus killing the cells and causing atrophy. The tissues are not uniformly affected. The earliest clinical manifestation is the deposit of iron in the anterior capsular cells of the lens where oval patches of the rusty deposit are arranged radially in a ring corresponding with the edge of the dilated pupil. This appearance is pathognomonic and leads eventually to the development of cataract (Plate XX, Fig. 1). The iris also becomes characteristically stained, first greenish and later reddish-brown. The vision of these eyes, however little affected by the primary injury, gradually fails owing to degenerative changes in the retina and lens. The retinal degeneration, associated with great attenuation of the blood vessels, eventually becomes generalized, taking the form of pigmentation resembling that of pigmentary retinal dystrophy. Secondary glaucoma of the chronic type is a common late complication; and, unless the foreign body becomes encapsulated or is removed in time, most of these eyes go blind.

Pathologically the deposits of iron are revealed by the Prussian blue reaction with Perls's micro-chemical stain. The characteristic blue pigmentation is found particularly in the corneal corpuscles, in the meshes of the trabeculæ, on the inner surface of the ciliary body, and in the retina where the whole retinal vascular system is clearly marked out. The anterior layers of the iris are impregnated, and in addition to subcapsular deposits in the lens, the fibres are also stained. There is always intense blue coloration immediately around the foreign body.

The reaction of *copper* or *brass* (as from percussion caps) varies with the content of pure copper. If the metal is relatively pure, a violent reaction ensues. Occasionally this results in the profuse

formation of fibrous tissue so that the particle becomes encapsulated ; more usually a suppurative reaction follows, which eventually retrogresses so that it results in shrinkage of the globe (Fig. 288).

If, however, the metal is heavily alloyed, a much milder reaction ensues—**chalcosis**. It becomes electrolytically dissociated and is deposited particularly where resistance to its migration is offered by continuous membranes. The typical sites for deposition are in the deeper parts of the cornea where it accumulates mostly at the periphery causing the appearance of a golden-brown Kayser-Fleischer ring (p. 221), under the capsule of the lens where it is deposited to form a brilliant golden-green sheen aggregated in radiating formations like the petals of a flower (*sun-flower cataract*) (Plate XX, Fig. 2), and occasionally on the retina at the posterior pole where lustrous golden plaques reflect the light with a metallic sheen. Not entering into chemical combination with the proteins of the cells as does iron, however, degenerative changes do not appear and the vision may remain indefinitely good.

Organized materials (wood and other vegetable matter) produce a proliferative reaction characterized by the formation of giant cells. Eyelashes may be carried into the anterior chamber in perforating wounds of the cornea, whether accidental or operative ; proliferation of the epithelium of the root hairs frequently leads to the formation of intra-ocular cysts. *Caterpillar hairs* may penetrate the eye (p. 173) exciting a severe iridocyclitis characterized by the formation of granulomatous nodules (*ophthalmia nodosa*).

The *diagnosis* of an intra-ocular foreign body is a matter of extreme importance, particularly as the patient is often unaware that a particle has entered the eye. In all suspicious cases a careful search must be made for a wound of entry ; and it may be very minute and difficult to find. If the particle has passed through the cornea, however, the most minute scar can always be seen in careful examination with the slit-lamp ; its detection in the sclera may be much more difficult or sometimes even impossible.

The anterior segment of the eye must be thoroughly explored with the loupe and slit-lamp and the angle of the anterior chamber with the gonioscope. A hole in the iris or an opaque track through the lens is pathognomonic. These tracks, together with the position of the wound of entrance, often give a valuable clue in localizing the foreign body. If the media are clear, the entire fundus must be similarly searched under full mydriasis.

Radiography is indispensable for the discovery and location of such foreign bodies as are radio-opaque. Fortunately these particles are usually metallic and many—although by no means all—can thus be demonstrated. Many methods of localization are available. One of the most useful—that exploited in the techniques of Mackenzie Davidson and Bromley in Britain, and Sweet and Dixon in America—depends on taking two stereoscopic pictures at two

fixed angles, and calculating the position of the foreign body from the displacement of its shadow with reference to a known opaque marker. This method is capable of great accuracy. A second method, of less accuracy, depends on taking successive photographs in various positions of rotation of the eye. A third method, developed largely by Comberg and also very accurate, relates the position of the foreign body to leaded markings on a contact glass inserted into the palpebral aperture.

A fourth method, the "bone-free" method introduced by Vogt, is of great value particularly in the case of material which is only poorly radio-opaque (glass, etc.). The anterior segment of the eye is photographed in two directions without the interposition of bone by placing a small dental film first in the inner canthus and then in the upper or lower fornix, and the position of the foreign body calculated from these. The posterior segment of the eye is similarly explored by inserting a similar film into Tenon's capsule and taking an oblique photograph (Fig. 289). The position of the foreign body relative to the sclera can be verified by taking subsequent exposures by the same technique after inserting a radio-opaque marker such as a minute needle into the sclera in its vicinity.

If properly applied most of these methods yield results of great accuracy; but such accuracy is necessary. When it is remembered that accuracy of localization of the order of 1 mm. is essential and that inaccuracy of this amount may lead to the needless sacrifice of an eye, it will be recognized how culpable is any carelessness in this respect.

A less accurate method of detection and localization is to exploit the alterations in a secondary induced current produced by a metallic particle in its vicinity. This principle has been incorporated in instruments (*locators*) wherein the searching element is a pointed probe and alterations in the current in the neighbourhood of the particle are revealed by the deflections of a dial or changes in the pitch of a loud speaker.

Treatment. A foreign body should be removed unless (1) it is inert and was probably sterile; (2) little damage has been done to vision; and (3) the process of removal will almost inevitably destroy sight. These conditions are most often fulfilled in the case of minute foreign bodies in the retina. Magnetizable foreign bodies are more easily removed than others since the use of the magnet considerably facilitates the technique.

Electro-magnets such as are used in ophthalmology utilize the principle of the solenoid wherein a coil wound around a central core renders the latter magnetic: this is fitted with suitably shaped terminals. The power of such a magnet depends on the electric current available (which is limited), the length and diameter of the wire in the coil and the mass of the core. The latter two are limited by the requirements of weight and manœuvrability. The effectiveness of any magnet depends on the magnetizability of the particle, its size which determines the number of lines of force which traverse it, and its distance away: the effectivity of a magnet diminishes as the cube of the distance from the particles to be attracted.

Intra-ocular foreign bodies are often lightly magnetizable and always small. *Hand magnets* (Fig. 290) which can readily be manipu-

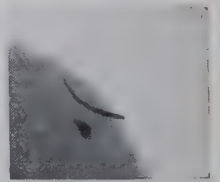


FIG. 289.
Bone-free radiography. A minute needle is inserted into the sclera near the presumptive position of the foreign body which is thus localized (Larsson).

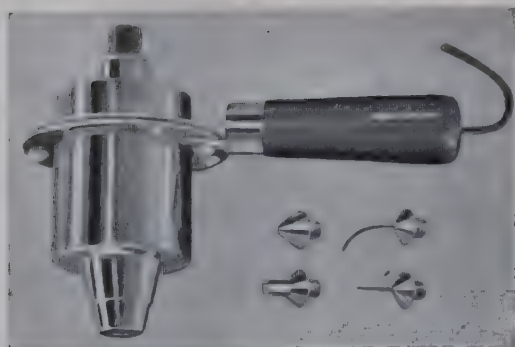


FIG. 290. Hand electro-magnet. Four small terminals are seen which screw into the point of the magnet (Hamblin).

adjusted at any angle with reference to the eye when the patient is recumbent (Fig. 291).

The *ring magnet* was deservedly popular before technical improvements allowed these conditions to be met. In it the coil is wound round a ring and an iron rod held in the centre of the ring becomes the terminal of the magnet: this the surgeon can manipulate with relative freedom when the patient is recumbent, his head being enclosed in the ring.

The time over which a magnet can be applied is limited by the amount of heat generated; prolonged applications can therefore only be made by repeated short exposures which are effective by gradually loosening a foreign body from an encapsulated bed or slowly drawing it through the tissues of the eye.

A *magnetic foreign body in the anterior chamber* is removed by the hand magnet through a keratome incision 3 mm. from the limbus, the keratome being removed moderately quickly but smoothly, so as to prevent loss of aqueous. The pole of the small magnet is then placed over the foreign body, outside the cornea, and moved towards the wound, thus drag-

lated by hand, are therefore only of value if their distance from the particle is small; they are only effective if virtual contact can be obtained. If the foreign body is more than 2 or 3 mm. away, a *giant magnet*, which derives its power from its bulk, must be used.

The latter are of two types. In the *Haab type of magnet*, the coil is wound round a central core. To be effective the instrument must be heavy and it should be exquisitely balanced and freely movable so that it can be



FIG. 291. Philps's giant electro-magnet (Hamblin).

ging the particle along the back of the cornea (Figs. 292 to 294). The posterior lip of the wound is then depressed with the pole and the foreign body is drawn towards it, out of the wound. If much aqueous is lost in the manipulations, the foreign particle is more likely to become entangled in the iris, and considerable difficulty may be experienced in freeing it. It is wise to wait some minutes for the anterior chamber to re-form before making a second attempt to remove it, and it may be necessary to pass a flat pole into the chamber. All the preparations for dealing with a prolapse of iris must be made (p. 415).

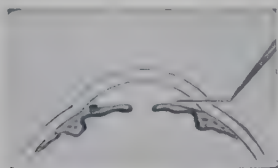


FIG. 292.

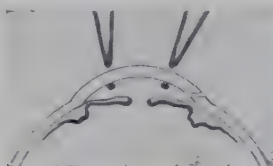


FIG. 293.



FIG. 294.

FIGS. 292-4. The removal of a magnetic foreign body from the anterior chamber. The keratome incision is made 3 mm. from the limbus in the segment opposite the site of the foreign body (Fig. 292). The foreign body is drawn across the anterior chamber by the hand magnet (Fig. 293) until it comes to the incision, out of which it is drawn (Fig. 294) (Philps).

If the foreign body is *on the iris* it frequently becomes entangled therein. In this event the iris must be drawn out of the wound and the part containing the foreign body abscised with de Wecker's scissors, as in the operation for iridectomy (p. 415).

If the foreign body lies within the lens, magnet-extraction is usually difficult; it is better to treat such particles as if they were non-magnetic (*vide infra*).

If a magnetic foreign body is in the vitreous or retina, the giant magnet is necessary for its removal. Two techniques are available—extraction by the anterior route whereby the foreign body is drawn round the lens and into the anterior chamber by the giant magnet and extracted therefrom by the hand magnet; and extraction by the posterior route whereby the particle is extracted directly through a scleral incision into the posterior segment of the globe. Formerly the anterior route was almost invariably employed owing to the fear of a retinal detachment following a posterior incision; this, however, can be obviated by diathermizing the site of operation. Undoubtedly less damage may be caused if the posterior route is used, particularly if the foreign body is large with jagged edges; moreover, the slight magnetizability of many alloys often makes the posterior route a necessity. If this technique is employed, however, accurate localization of the foreign body is essential so that it is removed by the shortest possible path.

In all cases, the extraction should be done as soon as possible after the injury. The longer the foreign body is left, the more firmly it becomes embedded in exudates or fibrous tissue, and the less the probability of its successful removal.

Extraction by the anterior route should be done under full regional analgesia, akinesia and mydriasis (p. 405). The terminal of the giant magnet is approximated to the cornea and the current switched off and on until the foreign body is drawn round the lens, enters the posterior chamber and is seen to bulge the iris (Fig. 295); at this point the



FIG. 295. The giant magnet in use for extraction by the anterior method (Philps).

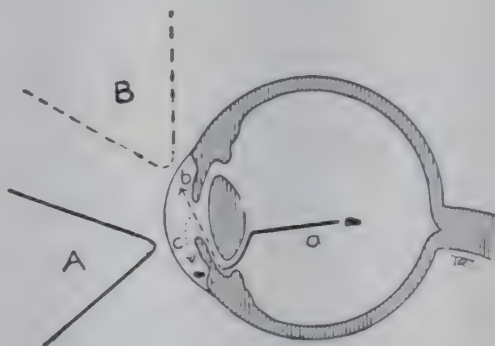


FIG. 296. The removal of a foreign body by the anterior route. A foreign body lies in the vitreous and with the giant magnet at position A it is drawn along the path *a* until it is seen to bulge the iris. The magnet is changed to position B and the particle is drawn through the pupillary aperture (*b*). The magnet is then switched off and the foreign body falls to the bottom of the anterior chamber (*c*).

current is immediately switched off lest the particle become entangled in this tissue. The terminal of the magnet is then approximated to the opposite point of the limbus so as to draw the particle between the iris and the lens through the pupil and into the anterior chamber (Fig. 296). Thereafter it is extracted in the manner already described.

Extraction by the posterior route is done through an incision as close to the foreign body as is practicable, as determined by previous localization; if the particle lies in the vitreous the incision is best made 8 or 9 mm. behind the limbus—the so-called “safe zone” in that it traverses the pars plana of the ciliary body, largely obviating any danger of immediate hæmorrhage or subsequent retinal detachment. The conjunctiva is reflected, the area around the incision diathermized as in the operation for retinal detachment (*q.v.*), the sclera carefully incised down to the choroid, the choroid and retina incised, and the point of the terminal of the magnet inserted into the lips of the wound but not into the cavity of the eye. If the foreign body is in the vitreous the giant magnet will be required; if it lies in the retina and localization has been accurate, the hand instrument suffices. The current is

then turned on; if the operation is successful the particle approaches and adheres to the terminal which is at once withdrawn, and after the terminal is free from the eye (not before), the current is switched off. The scleral wound is then closed by previously placed sutures.

The immediate effect of extraction of foreign bodies with the large magnet is often good, but irreparable damage is frequently done to the eye. The tracks through the vitreous may become filled with fibrous tissue; as this organizes and contracts, the retina is pulled up and total detachment destroys vision. Alternatively an iridocyclitis may be set up which may result in shrinkage or atrophic changes may develop.

The extraction of a non-magnetic foreign body from the anterior segment of the eye is often easy; from the posterior segment it presents great difficulties.



FIG. 297.

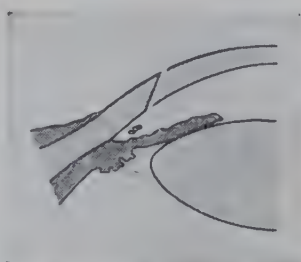


FIG. 298.

FIGS. 297-8. The removal of a non-magnetic foreign body from the anterior chamber. Fig. 297. The wrong incision. Fig. 298. The correct incision to allow access to the foreign body.

If the foreign body lies upon the iris it can usually be picked out by iris forceps through a suitably placed keratome incision. If it is entangled in this tissue, it is removed in the course of an iridectomy in the manner already described.

If it lies in the angle of the anterior chamber it is impossible to get at it with forceps by an ordinary keratome incision immediately over it (Fig. 297). The incision should be made at 3 mm. inside the limbus in the quadrant of the cornea lying over the foreign body, the point of the keratome being directed straight at it (Fig. 298). The foreign body can then be lifted out with toothless forceps, and the risk of prolapse of iris is also minimized.

If the foreign body is in the lens, a few days should be allowed to elapse for the aqueous to act upon the lens fibres. A curette evacuation (p. 425) is then performed and the foreign body will probably be evacuated with the lens matter, or it may be removed by forceps (or if magnetizable, by the small magnet). In a young subject it may be advisable to increase the opening in the capsule by discission a few days before doing a curette evacuation. In elderly patients it may be necessary to extract the lens by the operation for extraction of senile cataract (p. 425), but the large amount of soft lens matter will increase the dangers of the operation.

If a non-magnetizable foreign body lies in the posterior segment of the globe its removal is difficult. If it lies on the retina, and *if accurate localization has been attained*, preferably verified at the time of operation by bone-free radiography, it may sometimes be directly removed by forceps through an incision, preceded, as already described, by diathermy through the sclera immediately over the site. If it is in the vitreous and the media are clear, successful instances have been recorded of its removal by special forceps introduced through a similar incision and manipulated under ophthalmoscopic control. If this is impossible owing to the presence of a cataract, the same manœuvre has been accomplished through an illuminated *endoscope*, a small instrument designed on the same principle as the cystoscope. Such cases, however, have been rare and the technique is unusually difficult. Even if it is successful, inescapable damage may be done to the eye. As a general rule in such cases, the treatment lies between leaving the foreign body and watching the case or excising the eye if it has been badly traumatized.

The prognosis of the retention of a foreign body in the eye is always serious, even if little mechanical damage has been done at the time of injury. It varies with the site and the chemical nature of the particle. If the foreign body is allowed to remain and is inert, it may be retained indefinitely without affecting vision, although an iridocyclitis, sometimes appearing after many years, may be anticipated. If it is not inert and allowed to remain, the visual prognosis is bad. If the foreign body has been removed from the anterior chamber, the prognosis is usually good provided the lens was not wounded. A wound of the lens, however, gravely aggravates the position owing to the immediate difficulties of its evacuation, the subsequent irritative reaction, and the tendency for the development of secondary glaucoma. Even if the particle has been successfully extracted from the posterior segment of the globe, the subsequent history over a five- or ten-year period shows that many such eyes eventually lose useful vision from degenerative changes or retinal detachment, no matter how dramatically successful the operation had been.

SYMPATHETIC OPHTHALMITIS

Sympathetic ophthalmitis is the much dreaded condition in which serious inflammation attacks the sound eye after injury of the other. In recent years sympathetic ophthalmitis has become a rare disease, in spite of the fact that ophthalmic surgery has become more conservative. Although common in the American Civil and Franco-Prussian Wars, it was very rare during the two World Wars. This gratifying fact is due to increased skill in the treatment of perforating wounds, particularly in the application of antiseptic principles and improved medical organization which allowed early

treatment of such casualties. A perforating wound, however, is a source of great anxiety to the most experienced surgeon.

Sympathetic ophthalmitis almost always results from a perforating wound ; it is doubtful if this is aggravated by the presence of a foreign body which remains within the eye. Wounds in the ciliary region—the so-called “dangerous zone”—involving the ciliary body and leading to its incarceration in the scar, have always been considered especially dangerous ; it is doubtful if, *per se*, they are more dangerous than others. On the other hand, it is certain that wounds in which iris, ciliary body or lens capsule is incarcerated are more likely to set up sympathetic ophthalmitis than others. It is least liable to occur in otherwise likely cases if the wound or the retained foreign body is sterile. Moreover, it very rarely occurs if actual suppuration has taken place in the exciting eye. It is more likely to occur from retention of shot, a chip of stone, glass, china, etc., than from a particle of hot steel, probably because the latter is sterile ; perforating ulcers are very rarely a cause. It is also extremely rare without perforation, if indeed it ever occurs in these circumstances.

Children are particularly susceptible, but it occurs at any age. It usually begins four to eight weeks after the injury to the first eye (the exciting eye) has taken place, rarely earlier, but the onset has been reported as occurring nine days after the accident and may be delayed for many months or even years.

There is always iridocyclitis in the exciting eye. Usually it is a plastic iridocyclitis which has been set up by injury and has not subsided in the course of three or four weeks. Instead of quietening down the ciliary injection remains, there is lacrimation and the eye is tender : special attention should be directed to the presence or absence of precipitates (“k.p.”) on the back of the cornea. In the rarer cases of delayed sympathetic ophthalmitis the exciting eye has passed into a quiescent state and may have shrunk completely. The onset of sympathetic ophthalmitis in the second eye is then ushered in by return of irritation—ciliary injection, tenderness, etc.—in the shrunken globe. The exciting eye, while showing evident traces of old iridocyclitis, may yet possess useful vision.

Sympathetic ophthalmitis—the disease in the second or sympathizing eye—is almost always a plastic iridocyclitis differing clinically in no respect from this form of iridocyclitis due to other causes. In rare cases it manifests itself as a neuro-retinitis or choroiditis. In cases which the surgeon knows to be liable to the condition the first sign may be the presence of precipitates (“k.p.”) on the back of the cornea, noticed at this early stage because they have been anticipated. In other cases the patient first seeks advice for photophobia and lacrimation, or defective vision in the uninjured eye (sympathetic irritation).

Prodromal symptoms are sensitivity to light and transient

indistinctness of near objects due to weakness of accommodation. On examination at this stage there may be lacrimation, slight ciliary injection, tenderness of the eyeball, as shown by the patient shrinking from an attempt at examination, precipitates on the back of the cornea, and vitreous opacities ; occasionally there is some œdema of the optic disc. The prodromal symptoms may occur in intermittent attacks, spread over a considerable period.

When fully developed all the signs and symptoms of iridocyclitis are present, varying in degree according to the severity of the case. The prognosis as to vision is always doubtful, but if there is much deposition of plastic exudates in the pupillary area it becomes extremely grave. Cases showing little exudation ("serous iritis") but a deep anterior chamber and "k.p." have a more favourable prognosis, but they may at any moment develop into the severe plastic type. Tension, difficult to determine on account of tenderness, is moderately raised in the early stages. It may then pass into the condition of lowered tension with gradual shrinking of the globe ; or the iridocyclitis may subside, the eye quietening down and retaining fair vision. In the worst cases a ring synechia forms and secondary glaucoma supervenes (p. 230), or both *occlusio* and *seclusio pupillæ* or a total posterior synechia (p. 230) occurs and the eye shrinks. Sympathetic ophthalmitis sometimes takes two or more years to run its course but the disease is self-limited.

Pathologically the microscopic features in both the exciting and the sympathizing eye are the same. In the earliest stages, examination shows nodular aggregations of lymphocytes and plasma cells scattered throughout the uveal tract. The pigment epithelium of the iris and ciliary body proliferates to form nodular aggregations (Dalén-Fuchs nodules) and the tissues become invaded by lymphocytes and epithelioid cells. The retina also becomes heavily infiltrated especially in the neighbourhood of the vessels. In the later stages the infiltrate becomes diffuse and giant cells appear ; in fact, the condition is scarcely distinguishable from tuberculosis of the uveal tract, although caseation is never present. These are merely the signs of reaction to a relatively mild form of irritation.

The *ætiology* of the condition is unknown, but two theories usually receive preference—that it is infective or allergic in nature. Since no organismal invader has been demonstrated, a hypothetical virus infection is often inculpated which, like many other viruses, may have the habit of remaining long quiescent. The view that the infection may travel from one eye to the other by the optic nerves and chiasma has no substantial evidence to support it ; it would seem more probable, if a virus exists, that the organism is harmless to other organs of the body and finds a suitable nidus only in the other eye, perhaps owing to allergic hypersensitivity of the uveal tract.

On the other hand, a purely allergic origin to uveal pigment has been postulated. Uveal pigment can act as an allergen and sufferers from sympathetic diseases may show a skin sensitivity to it. Perhaps the truth lies in a combination of both views.

The *treatment* of sympathetic ophthalmitis is one of the most difficult problems in ophthalmology, and often demands the exercise of great judgment.

It is, in the first place, prophylactic. In every case of perforating wound, with or without the retention of a foreign body, the question of excision of the eye on account of danger to its fellow arises. It may be assumed as an axiom that *sympathetic ophthalmitis never occurs after the excision of an injured eye unless it has already commenced at the time of operation*. Hence, early excision is a positive safeguard against the disease. The rule should be to excise any eye which is so injured that it is improbable that useful vision will be regained. In cases wherein this is doubtful, expectant treatment, including the topical administration of steroids, may be adopted for a time. If the eye quietens down quickly it is unlikely to set up sympathetic inflammation. The chief causes which keep up irritation are entanglement of the iris or ciliary body or lens capsule in the wound ; every effort must therefore be made to obviate this.

During this expectant period if, despite treatment, the eye continues to be irritable, with ciliary injection, photophobia, and lacrimation, and above all if "k.p." appear, the eye should be excised. It is seldom wise to wait longer than a fortnight unless there are undoubted signs of amelioration. It must be remembered that children are more susceptible to the disease than adults.

Even more difficult to decide is the course to adopt in those cases in which sympathetic ophthalmitis has already supervened. If the case is seen early, shortly after the onset of inflammation in the sympathizing eye, and if the injured or exciting eye has no useful vision, this useless eye should be excised at once. There is no question that the excision of the exciting eye has a good effect upon the process in the sympathizing eye if performed early. At a later stage there is no evidence to show that it exerts any influence.

The chief difficulty arises when the exciting eye has useful vision and the inflammation in the sympathizing eye is severe. If this is the condition soon after the onset it may be wise to excise the injured eye. If, however, a considerable time has elapsed since the injury, excision of the exciting eye is likely to have little or no influence upon the process. Moreover, in the end the injured eye may have better vision than the sympathizing one, for if the iridocyclitis is severe the sympathizing eye may be lost in spite of all efforts. In these conditions, therefore, the injured eye should be retained.

The treatment of sympathetic iridocyclitis is that of iridocyclitis in general (p. 241) with the proviso that cortisone preparations have a more dramatic effect than in most other ocular inflammations. At the earliest suggestion of inflammation, steroids should be given intensively (p. 145) ; subconjunctival injections reinforced by the ointment are the most effective method. In

advanced and desperate cases, it may be introduced into the anterior chamber. Intravenous ACTH may be valuable at the commencement of treatment. In all cases the treatment should be continued for some time lest relapses follow its cessation, and the eye should be watched over a period of many months. If the immediate crisis can be tided over in this way and recurrences prevented, comfort may be derived from the fact that the disease is self-limiting. The use of steroids has completely altered the prognosis of this disease *if such treatment is commenced early*. If, however, the inflammation has taken a firm hold and the uvea is heavily infiltrated, the outlook is much less bright.

In cases which have run their course and suffered severe organic changes, improvement of vision may occasionally be obtained by operation, but no such interference is to be contemplated until the eye has been quiet for many months. In the milder cases an optical iridectomy may do good. In the worst cases, so long as there is perception and moderately good projection of light, more desperate operations such as extraction of the lens may be justifiably undertaken if the other eye is blind or has been removed.

CHAPTER 27

OPERATIONS UPON THE EYEBALL

IN this chapter the more important operations only will be noted. Of these the commoner classical procedures will be described in some detail ; only the general principles of more specialized techniques will be indicated.

PRELIMINARY MEASURES

Before intra-ocular operations, particularly major operations such as cataract extraction, *a general survey* of the patient is advisable which should include an investigation of the blood-pressure and the general cardio-vascular condition and of the urine to exclude the presence of sugar and albumin. Few general diseases are a complete contra-indication to an ocular operation ; but it is well to be forewarned of possible complications. A search for focal sepsis, such as abscessed teeth or enclosed pus in a nasal sinus, should also be made, and if practicable these should be cleared up. Caution should, however, be exercised, particularly in aged or frail patients ; if radical treatment seems inadvisable, operation under the “ umbrella ” of a suitable antibiotic should be considered.

Local bacteriological precautions should also be taken by the examination of a preliminary swab and suitable antibiotic treatment given if any infection is present in the conjunctival sac. Dacryocystitis should be eliminated—a nasal-drainage operation may be performed, the sac may be excised if surgery is more urgent or, in an emergency, the puncta, both upper and lower, occluded by a diathermic needle (p. 516).

ANÆSTHESIA FOR EYE OPERATIONS

Most ophthalmic operations can well be conducted under local analgesia, preferably with premedication. While the co-operation of the patient is always desirable, it is not always given, and it is unfair in the trying circumstances of an operation to expect that it should be ; but it is easy by adequate basal sedation, analgesia and akinesia to ensure that events will not get out of hand. In the case of neurotic and highly strung patients a general anæsthetic may be used.

Local Analgesia. (1) *Surface Analgesia.* Cocaine and its many derivatives are readily absorbed by the conjunctiva and cornea, and produce complete analgesia after the instillation of 4 drops of a 2 per cent. solution at intervals of three minutes. The iris, however, is not rendered completely analgesic by this method.

Cocaine occasionally causes alarming symptoms in old and debilitated patients and as an idiosyncrasy, but this is very rare : as a precaution, sustained pressure over the lacrimal sac during instillation will prevent absorption from the nasal mucous membrane. One of its many substitutes is preferable since these neither damage the corneal epithelium nor do they dilate the pupil.

(2) *Infiltration and Regional Analgesia.* Infiltration analgesia with procaine or a suitable substitute is employed to effect analgesia of the iris. A retro-ocular injection may be used to infiltrate the ciliary ganglion and the surrounding tissues : 1 ml. of procaine (4 per cent.) is injected with a fine needle, 5 cm. long, passed through the skin of the lower lid at the outer and lower angle of the orbit and directed towards the apex of the orbit for a distance of 3.5 cm. Occasionally a large vein is punctured giving rise to a proptosis sufficient to delay an intra-ocular operation for a few days. An alternative is to make a similar injection into Tenon's capsule just behind the equator about half-way between the temporal border of the superior rectus and the upper edge of the lateral rectus. A fine needle is used, and care is taken to insert it very obliquely through the conjunctiva, to avoid the site of a vortex vein, and to keep close to the sclera.

In cases wherein a general anæsthetic is undesirable the eye can be removed painlessly with an injection of 6 ml. of procaine and adrenaline into the apex of the orbit.

Pre-medication. It is usually wise to administer sedatives before operation to allay the natural fear and apprehension of the patient. For this purpose several analgesic and hypnotic drugs may be employed such as a mild barbiturate combined with a morphine preparation such as Pethidine (50 to 150 mg. intramuscularly). It is a good thing to give the same drug as a sedative the night before, partly to ensure a good sleep and partly to make sure that no unpleasant reactions arise since the effect of all these drugs varies considerably among individuals.

General Anæsthesia. *Basal anæsthesia by the intravenous administration of drugs* is peculiarly suited to ophthalmic surgery. A useful drug for this purpose is pentothal sodium, a rapidly acting barbiturate which produces a fall of blood pressure and intra-ocular pressure, good relaxation and moderately quick recovery of consciousness. A single injection suffices for an operation of ten to twenty minutes' duration. For longer operations more of the solution is periodically injected. Conjunctival and corneal reflexes are abolished, but it is wise to use local analgesia and akinesia in addition. During anæsthesia the eyes are central and directed slightly upwards.

Inhalation anæsthesia is seldom indicated unless in children when the operation is prolonged. Its chief disadvantages are the deviation of the eyes, vascular congestion, and post-operative restlessness and vomiting.

When much hæmorrhage is expected (closed-angle glaucoma) it may sometimes be obviated by the injection of sympatholytic drugs such as hexamethonium which act by lowering the general blood pressure. They should, however, be employed only by an expert and with great care. Serious accidents due to cerebral or retinal anoxia may occur entailing risks which, although rare, are not justified merely to make the surgeon's task easier by the attainment of a bloodless field of operation.

AKINESIA FOR EYE OPERATIONS

In intra-ocular operations involving a wide opening of the globe (e.g., cataract) temporary paralysis of the orbicularis muscle is advisable in order to prevent squeezing together of the lids.

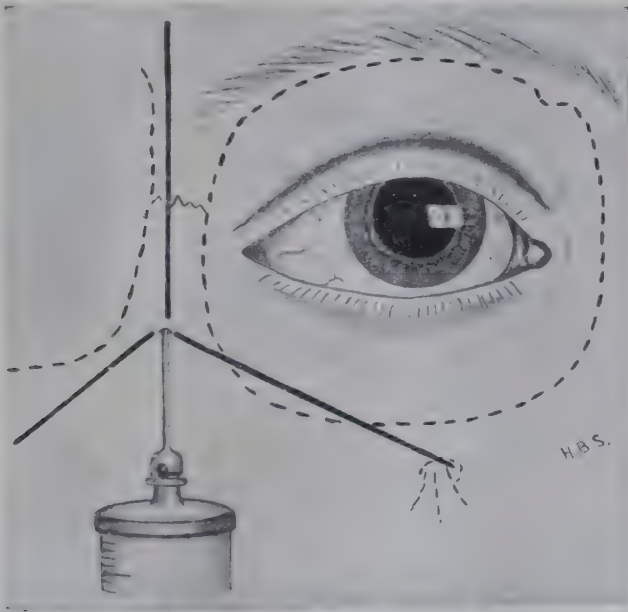


FIG. 299.

For this purpose a *facial block* may be employed. Four to 5 ml. of procaine are injected down to the periosteum covering the neck of the mandible where the upper branches of the facial nerve pass forwards and upwards. The patient should be instructed to open his mouth, and the position of the condyle and temporo-mandibular joint is located by the operator's left forefinger. After closing the jaw the needle is inserted at a point half an inch below the position of the condyle and passed straight down to the periosteum where the solution is injected, and firm pressure and local massage are applied. Paralysis of the orbicularis rapidly follows.

Alternatively the branches of the facial nerve may be blocked as they run over the malar bone. A needle is inserted down to the periosteum of the malar bone at a point about 1 cm. below and behind the outer canthus and passed upwards towards the temporal fossa, forwards and downwards towards the infra-orbital foramen, and downwards and backwards towards the tragus for an inch or so (Fig. 299).

When the lids have been paralysed in this way, post-operative closure should be ensured either by filling the conjunctival sac with an antibiotic ointment and carefully closing the lids or by inserting a stitch into the skin of the upper lid 3 mm. above its margin. This is fixed to the cheek by adhesive plaster after the operation and removed at the first dressing.

Fixation of the globe by a stitch passed through the tendon of the superior rectus muscle is necessary in intra-ocular operations conducted under general anæsthesia and is advisable in all intra-ocular operations wherein looking down is essential. It is more effective after the injection of 0.5 ml. procaine into the belly of the muscle. During the operation the stitch is clamped without tension to the head-towels by artery forceps (Fig. 350).

Akinesia affecting particularly the extra-ocular muscles can be attained by the systemic injection of curare-like preparations such as *d*-tubocurarine chloride (5 to 9 mg. intravenously). Small doses only are required since these muscles are the most susceptible in the body to the action of these relaxants, but they should be used with care and only by an expert.

The Speculum. In most operations a simple speculum (Lang's Fig. 300) is adequate. Many others are available which are supported on the orbit but the simplest and safest method is to employ lid-stiches.



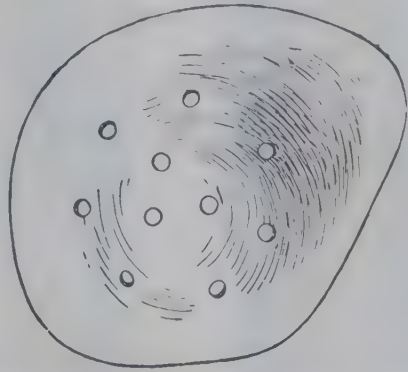
FIG. 300. Lang's speculum
(half size).

The upper lid stitch (*vide supra*) instead of being inserted in the middle is passed through the skin twice at the junctions of the middle third with the outer and inner thirds, and it, as well as the central superior rectus stitch, is clamped on the head-towels to keep the upper lid back. A skin stitch in the lower lid depresses it by the weight of an artery forceps hanging down over the cheek.

General Routine. The preparation of the eye immediately before the operation is the same in all cases. The eye is irrigated with normal saline solution. The skin of the lids is dried and painted with a detergent solution or spirit. Two or three drops of an antibiotic are instilled. Five drops of a cocaine substitute are instilled at three-minute intervals and one drop into the other eye to diminish the risk of closure of the lids. The orbicularis oculi is paralysed; an injection of procaine is made into Tenon's capsule or the ciliary ganglion if necessary. Sterile towels are draped round the head,

neck and chest and the face covered with a mask in which an aperture is cut to give access to the eye. The lid stitching is inserted after a minute local injection of procaine into the skin. The speculum (if employed) is inserted and the eye is ready for operation.

FIG. 301. "Cartella" shield for right eye.



In most operations *fixation of the eye* is required. This is usually attained by fixation forceps (Fig. 303) which should take a deep grip of the conjunctiva so as to include the episcleral tissue or an insertion of one of the muscles; otherwise they are liable to tear the conjunctiva if much traction is exerted, as by an involuntary movement of the patient. The eye is then pulled gently forwards; the forceps should never be pressed backwards, particularly in operations wherein the globe is opened, lest the pressure expel some

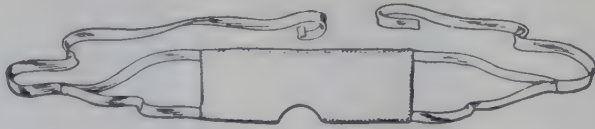


FIG. 302. Diagram of Moorfield's cataract bandage. The semicircular opening is for the bridge of the nose. The upper and lower tapes are passed above and below the ears. The long single tapes pass behind the head and are brought forward and tied over a pad of wool placed at the upper part of the bandage over the forehead.

of the intra-ocular contents. As an alternative in some operations a suture may be inserted at a convenient site into the subconjunctival tissue and immobility in a particular direction attained by gentle but firm traction thereon.

The routine *post-operative treatment* is the instillation of sterile atropine (or other drops as required), the insertion of antibiotic ointment, the application of a light gauze pad and bandage—and to bed. If desired, the gauze pad may be soaked in sterile paraffin and covered by cotton-wool moistened with normal saline. When the globe has been widely opened, protection against accidental injury may be obtained by a rigid cover shaped to the orbital rim; a cartella shield of stiff cardboard is suitable (Fig. 301). This and

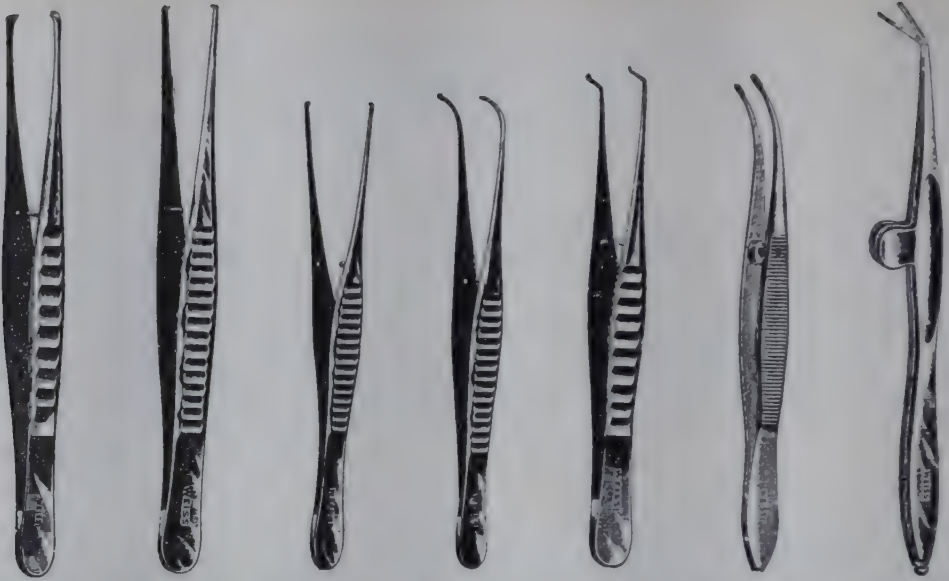


FIG. 303. FIG. 304. FIG. 305. FIG. 306. FIG. 307. FIG. 308. FIG. 309.



FIG. 310.

FIG. 311.

FIG. 312.

FIG. 313.

FIGS. 303-13. Ophthalmic instruments (half size) (Weiss).
(Fig. 311, two-thirds size.)

Fig. 303, Fixation forceps ; Fig. 304, Conjunctival forceps ; Fig. 305, Iris forceps (straight) ; Fig. 306, Iris forceps (curved) ; Fig. 307, Disc forceps ; Fig. 308, Capsule forceps ; Fig. 309, de Wecker's scissors ; Fig. 310, Needle holder ; Fig. 311, Spring scissors ; Fig. 312, Blunt-pointed scissors ; Fig. 313, Sharp-pointed scissors.



FIG. 314.

FIG. 315.

FIG. 316.

FIG. 317.

FIG. 318.

FIG. 319.



FIG. 320. FIG. 321.

FIG. 322.

FIG. 323.

FIG. 324.

FIG. 325.

FIG. 326.



FIG. 327.

FIG. 328.

FIG. 329.

FIG. 330.

FIG. 331.

FIG. 332.

FIGS. 314-32. Ophthalmic instruments (Weiss). (Figs. 314-26, two-thirds size ; Figs. 327-32, three-quarters size.)

Fig. 314, Broad Graefe knife ; Fig. 315, Narrow Graefe knife ; Fig. 316, Straight keratome ; Fig. 317, Angled keratome ; Fig. 318, Broad needle ; Fig. 319, Paracentesis needle ; Fig. 320, Cystitome ; Fig. 321, Curette ; Fig. 322, Vectis ; Fig. 323, Ziegler's needle ; Fig. 324, Straight needle ; Fig. 325, Expression hook ; Fig. 326, Strabismus hook ; Fig. 327, Tooke's knife ; Fig. 328, Franceschetti's corneal trephine ; Fig. 329, Corneo-scleral trephine ; Fig. 330, Iris hook ; Fig. 331, Iris repositor ; Fig. 332, Cyclodialysis spatula.

the pad can be secured in place by plastic strapping or adhesive plaster. When both eyes are covered a many-tailed or "Moorfields" bandage (Fig. 302) saves manipulation in dressing and is cooler and lighter. For the first few days in bed a semi-sitting (Fowler's) position is very suitable particularly in intra-ocular operations in which the wound traverses the upper part of the limbus.

Most ophthalmic operations are performed with surgical cutting instruments; three techniques, however, should be noted which have special indications.

Operations with Diathermy

Diathermy is used in ocular surgery in the following circumstances: (1) to excite a plastic choroiditis to cause adhesions between the retina and choroid and close a retinal hole in retinal detachment, or as a prophylactic measure to prevent its occurrence in cases in which thin and



FIG. 333.



FIG. 334.

FIGS. 333-4. Diathermy terminals. Fig. 333, Blunt; Fig. 334, Perforating needles.

degenerate areas occur in the retina, and before opening the posterior segment of the globe as for the removal of a foreign body; (2) in glaucoma to cause an atrophy of the ciliary body; (3) to destroy intra-ocular inflammatory lesions (retinal periphlebitis) or neoplasms, either simple (as an angioma) or malignant, as an alternative to radiation when excision of the eye is contra-indicated.

In operations with diathermy through the sclera the inactive electrode is bandaged onto the patient's arm or leg; it is usually a metallic band to which contact is made by smearing the two surfaces with a salt-containing paste. The active electrode is applied to the eye by one of two methods. In *surface diathermy* a blunt terminal is laid on the surface of the globe; in *perforating diathermy* the terminal takes the form of one or more minute sharp needles thrust through or partly through the ocular coats (Figs. 333-4). In each case the current should be individually regulated. In surface diathermy a grey burn should be left on the sclera and immediate ophthalmoscopic inspection should reveal a grey or white coagulated area some 2 mm. in diameter in the fundus. An average application involves the use of a current of 80 milliamperes for seven seconds, but since individuals vary much in their resistance, subsequent applications should be modified according to the effect of the first. In perforating diathermy about 40 milliamperes are usually used for three seconds; the needle should traverse the ocular coats with little resistance, lightly charring the tissues on its way, and the ophthalmo-

scopic appearance should be similar. In this case the area of reaction is slightly smaller so that more applications are necessary.

Light-coagulation is a useful technique wherein a chorioretinal burn is caused by a brilliant beam of light derived from a specially designed Xenon arc-lamp. The apparatus is elaborate and allows an ophthalmoscopic view of the retinal lesion. It may be used in cases of retinal detachment to coagulate the region of the tear in the central area of the fundus, provided the retina is not far removed from the underlying choroid. It may also be used to destroy a small neoplasm or inflammatory lesion in this region of the fundus or on the iris. A smaller lesion of the same type can be made by a *laser* incorporated into an ophthalmoscope.

Cryosurgery is a technique used to injure tissues by the application of intense cold. This is achieved by a cryoprobe, a pencil-like instrument held by the surgeon, cooled to a temperature varying from -40°C to -100°C by solid carbon dioxide or liquid nitrogen (Fig. 335). The method is useful in such procedures as the surgery of cataract and retinal detachment.

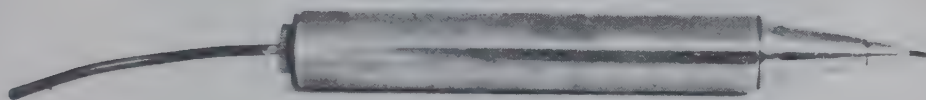


FIG. 335. A cryoprobe.

OPERATIONS ON THE CORNEA

Paracentesis may be employed for hypopyon ulcers, hyphæma with raised tension or occasionally for hypertensive cyclitis. As a rule the paracentesis is performed near the periphery of the cornea

The eye is steadied with fixation forceps and an incision made approximately 2 mm. within the limbus at the point selected. Generally the temporal limbus is most convenient ; for the evacuation of a hypopyon the lower limbus is used. A keratome (Fig. 316), a broad needle (Fig. 318) or a paracentesis needle (Fig. 319) may be employed. This is inserted in the plane of the iris to make a valvular opening, and directly the point traverses the cornea the plane of the blade is altered so that it lies close to the inner surface of this tissue to avoid wounding the iris or lens. It is then pushed inwards until the incision is sufficiently long, and carefully withdrawn so that little or no aqueous escapes. A sudden withdrawal with loss of much aqueous may lead to hæmorrhage or a prolapse of the iris ; the means to deal with this should therefore be at hand. An iris reposer (Fig. 331) is then inserted and the peripheral lip of the wound slightly depressed so that the aqueous escapes slowly with a minimum of disturbance. Such an incision can be repeatedly re-opened on successive days by the iris reposer.

In corneal ulcers a paracentesis may be made in the floor of the ulcer itself. The same technique is employed, the blade of the instrument being inserted at an angle of 45 degrees with the cornea until it enters the anterior chamber where it is at once altered so as to lie against the back of the cornea to avoid injury to the lens. In very severe ulcers an incision may be made transfixing the cornea from within outwards through the ulcer itself—Saemisch section.

In *Saemisch Section* (an operation, despite its name, devised by Guthrie) the point of a narrow Graefe knife (Fig. 315) is inserted in healthy cornea outside the edge of the ulcer. The edge of the knife is directed forwards, so that if the lens advances before the section is complete it will touch the back of the knife and not be wounded. The knife is passed across the anterior chamber until the point is seen beyond the opposite edge of the ulcer. The counter-puncture is made in healthy cornea (if possible) and the knife is pushed on so as to cut out. The aqueous pours out and carries with it much of the hypopyon. The operation is best done under a general anæsthetic since when the iris comes suddenly into contact with the cornea the pain is severe.

Keratoplasty. The general principles of corneal grafting are as follows. In *perforating keratoplasty* a disc in the centre of the cornea is involved. From the donor eye, usually a cadaver eye preserved by refrigeration, the cornea is removed and a disc of the required diameter is punched out with the smaller of two paired keratoplasty trephines (Fig. 328) and transferred to a watch-glass containing saline. With the larger of the two trephines a corresponding disc is then marked on the recipient cornea and double-armed fixation sutures inserted, either at the limbus so that they cross above the graft or in the cornea around the disc so that the graft may be secured by direct suturing. The trephining is then continued through the thickness of the cornea until aqueous begins to escape; the disc, including any tags of Descemet's membrane, is then completely removed by a specially designed scissors or knife. The graft is then carefully laid upon its bed and the sutures tied. Full mydriasis should be maintained after operation to prevent anterior synechiæ. Sutures are removed in about 14 days until which time both eyes are bandaged.

In *lamellar keratoplasty* a trephine of the Franceschetti type (Fig. 328) is used fitted with a guard so that the cut reaches only a desired depth. A disc of the required depth (usually about 0.4 mm.) is cut in the donor's cornea with the trephine and the lamella removed with a special corneal knife. A similar procedure is performed on the recipient's eye and sutures inserted as for penetrating keratoplasty. The graft is then inserted into its bed and the sutures tied. The eye should remain undisturbed for four days.

Tarsorrhaphy, although not strictly a corneal operation, is performed for corneal conditions, particularly neuroparalytic or exposure keratitis. The operation may aim at more or less total closure of the lids or alternatively be confined to one half, usually the external, so that the palpebral aperture is narrowed rather than

closed. Apart from the placement of the sutures both operations follow the same technique.

The mucous membrane is dissected up from the margin of the lower lid just posterior to the grey line (p. 483) over rectangular areas about 6 mm. long on each side of the middle of the lid. The edge of the upper lid is similarly treated at the corresponding positions. Two mattress sutures are then passed through rubber sheeting and the skin so that they come out at the posterior edge of the bare surface (Fig. 336)—not on the posterior surface of the lid where they would rub against the cornea. After being similarly carried through the skin of the other lid and the rubber they are tied, the freshened surfaces being brought into contact. In a few days the lids will be firmly adherent. Alternatively, a continuous subcuticular suture along the raw lid-margin may be used.

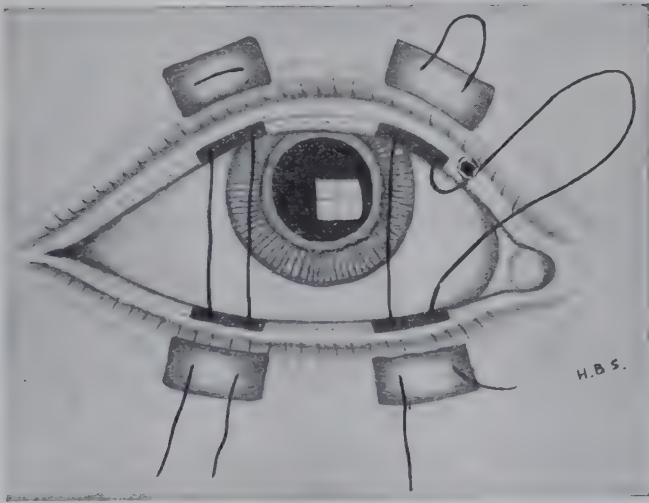


FIG. 336. Tarsorrhaphy.

OPERATIONS UPON THE IRIS

Iridectomy, which consists of the abscission of a portion of the iris, is performed for the following conditions :—(1) Prolapsed iris ; (2) corneal or lenticular opacities (optical iridectomy) indicated only when the opacities are localized and stationary and there is good perception and accurate projection of light ; (3) closed-angle glaucoma ; (4) as part of cataract extraction ; (5) threatening ring synechia ; (6) foreign bodies in, or small cysts or tumours of the iris.

Iridectomy for Prolapsed Iris. The eye is steadied by fixation forceps or a fixation suture and the prolapsed portion of the iris pulled outwards and from side to side with a view to freeing any adhesion. If consolidation has occurred it may be impossible to free it, and in this case effectual iridectomy cannot be performed. Having freed the iris as much as possible, the fixation forceps are handed over to an assistant. The prolapse is seized with iris forceps

held in the right hand, as close to the cornea as possible, and drawn well out from the wound. The second pair of iris forceps, held in the left hand, is then applied again as near the cornea as possible, and the iris drawn still further out. (Toothed capsule forceps (Fig. 307) are very good for this purpose.) de Wecker's scissors (Fig. 309) are then taken in the right hand and the iris is cut off close to the cornea (Fig. 337). If the operation has been successfully performed the stumps of iris retract into the anterior chamber and are free from the wound. Atropine is instilled and a pad and bandage applied.

It is to be noted here that the iris is extremely ductile; it can be dragged out much farther than might be expected, and it must be dragged out as far as possible so that the incision is through clean iris tissue, all the soiled part being removed. Too sudden jerks have been known to drag the whole iris out of the eye, since it tears away at the thinnest part, near the ciliary border.

Freeing of the iris is especially difficult with quasi-tangential corneal wounds. In such cases it is a good plan, if there is an anterior chamber, first to make a small keratome incision in the cornea on the opposite side to the wound, 3 mm. inside the limbus (Fig. 337). After the prolapse has been abscised in the usual manner a repositor can be passed through this incision and swept over the surface of the iris. This manœuvre is useful for freeing the iris from a punctured wound near the limbus, in which incarceration occurs but is so small as to make abscission through the original wound impracticable. In some cases it is advisable to cover the wound with a conjunctival flap. Atropine should always be instilled (never eserine).

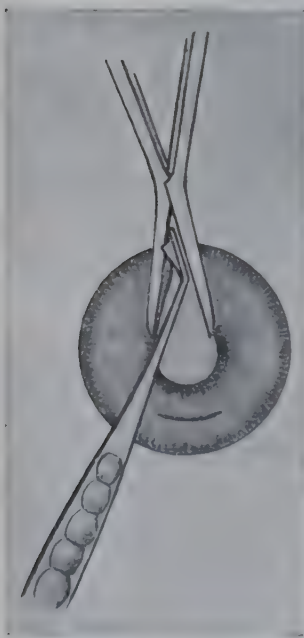


FIG. 337.

An optical iridectomy should be as narrow as possible in order to avoid dazzling and to obtain an approximation to stenopæic vision (vision through a narrow slit, p. 56). It should not extend to the ciliary border. The site of election is down and in (Fig. 338), but in the case of corneal opacities the clearest region of the cornea must be chosen, unless this happens to be above, in

which case the coloboma would be covered by the lid and useless for vision.

A keratome (Fig. 316) is inserted at or just inside the apparent corneo-scleral margin, and withdrawn as described in the operation of paracentesis (*q.v.*). The iris forceps (Fig. 306) are inserted closed, then opened very slightly and the iris seized just outside the pupillary margin: or the iris may be drawn out with a blunt iris hook (Fig. 330)

which allows a narrower coloboma to be made. The iris is drawn out of the wound and a portion excised with de Wecker's scissors (Fig. 309). A slit-like coloboma is made by holding the scissors so that the blades are in the direction of a radius of the iris (Fig. 340).

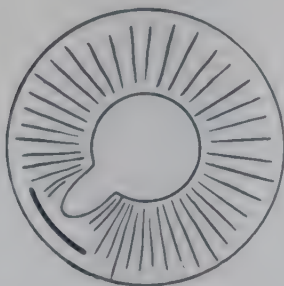


FIG. 338. Diagram of wound and coloboma in optical iridectomy at the site of election.

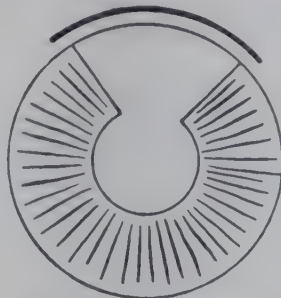


FIG. 339. Diagram of wound and coloboma in a sector iridectomy.

Iridectomy for Iritis (p. 242) should be performed as in optical iridectomy, but preferably in the upper part of the iris, and in this operation the coloboma should be wider.



FIG. 340.

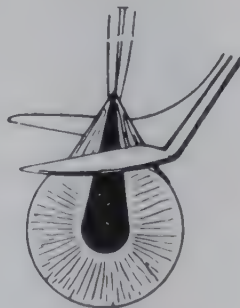


FIG. 341.

FIG. 340. Iridectomy with blades of de Wecker's scissors held radially. FIG. 341. The blades held at right angles to the previous position. The former method results in a narrower coloboma, such as is preferable in optical iridectomy and in extraction of senile cataract with iridectomy

Iridectomy in Combined Extraction of Cataract. See p. 428.

Iridectomy for Closed-angle Glaucoma may be peripheral ("button-hole") or total. In the first case, the operation is designed to re-establish communication between the posterior and anterior chambers; the second is rarely required.

In acute exacerbations of closed-angle glaucoma a general anæsthetic or deep regional analgesia is required, chiefly because the high tension prevents sufficient absorption of a local anæsthetic

to render the cornea—much less the iris—insensitive, partly also because the patient's self-control has been shattered by pain and anxiety. Control of the eye by a superior rectus stitch is also advisable. A miotic should always be instilled into the unaffected eye.

In the classical operation the incision is made by a narrow Graefe knife (Fig. 315) as for a cataract extraction (*q.v.*) except that the section is smaller and is kept 2 mm. behind the apparent corneo-scleral margin. An easier and safer procedure is the *ab externo incision*. A conjunctival flap is cut 3 mm. from the limbus from 2 to 10 o'clock. An incision is then made 2 mm. behind the corneo-scleral margin from 12.30 to 11.30 o'clock by a few strokes of the blade of a Bard-Parker knife to open the anterior chamber. The lens cannot be wounded since this opening is peripheral to its equator. Since rapidity in the re-formation of the anterior chamber avoids the formation of post-operative peripheral synechiæ, it is well to introduce corneo-scleral sutures (including the conjunctiva) when the incision is half-way through the sclera (p. 428). When the incision enters the anterior chamber the iris presents in the wound or will do so on slight pressure on the cornea.

In a *peripheral iridectomy* the eye is gently rotated downwards by the superior rectus stitch, the conjunctival flap is turned down with a sterile swab and the surgeon, taking the iris forceps in his left hand and de Wecker's scissors in his right, abscises a portion of the iris near its base leaving the sphincter intact.

In a *complete (sector) iridectomy*, the surgeon inserts the points of the closed forceps in the wound, carrying them to a position half-way between the pupillary and ciliary margins of the iris, slightly to the right of the vertical meridian of the cornea. The forceps are opened and the iris is gripped, pulled out and cut to the right side of the forceps. This stroke makes the right limb of the coloboma. The iris is then drawn across towards the left and torn from its attachment for the whole width of the section. It is then drawn a little back towards the right, so that it may not be jammed into the left angle of the incision and the free part cut off by a second snip of the scissors (Fig. 339).

The tip of the iris repositor is then introduced into the wound and insinuated between the cornea and the iris on both sides, and by radially directed movements the iris is smoothed out towards the centre of the pupil so that if the edges of the coloboma are caught in the angle of the wound they will be freed. Only when the edge of the pupil is in its natural position and looks circular but for the defect in the upper part is the surgeon convinced that the pillars of the coloboma are free from entanglement in the wound. The scleral suture is now tied so as to close the conjunctival incision at the same time. Atropine is generally instilled and the eye bandaged.

The chief complications which may arise during the operation are hæmorrhage into the anterior chamber (not usually serious, but inconvenient) ; wounding of the lens (often not discovered until opacity develops) ; and severe intra-ocular hæmorrhage.

Iridotomy is section of the iris without the abscission of any portion. It is employed for making a new pupil when the normal pupil is closed or has been drawn up to the wound of a faulty cataract extraction with incarceration of the pillars of the coloboma (Fig. 342).

A keratome incision, 3 or 4 mm. long, is made near the periphery of the cornea at the most suitable part, usually the temporal side. The direction of the section should correspond with the position of the proposed puncture in the iris, i.e., it will be approximately radial. This facilitates the opening and shutting of the iris scissors, and minimizes the bruising of the lips of the wound. The closed blades, one of which is pointed, of de Wecker's scissors (Fig. 309) are passed into the anterior chamber so that the pointed blade is forced through the iris and passed on horizontally. The blades are then closed, a horizontal slit being made in the iris. This cuts across the stretched fibres, which retract, leaving an oval artificial pupil.

Sometimes the iris can be hooked out through a keratome incision by means of an iris hook (Fig. 330), and a piece cut off. This gives a good pupil, but is, of course, strictly speaking an iridectomy.

The results of iridotomy for artificial pupil are often disappointing, the inflammatory reaction causing the gap to fill with exudate which organizes into scar tissue. It is, however, remarkable how little reaction follows in some cases. In others, particularly aphakic eyes, the organization of the tissues may be too dense to permit the ordinary operation. In this event, after complete akinesia and pre-placing corneo-scleral sutures (p. 428), a large corneal section is made as for cataract (*q.v.*), the cornea retroflexed by traction on the suture, and a triangular piece of iris more deliberately abscised. The sutures are then rapidly tied.

Iridotomy may be a necessary preliminary to iridectomy in cases of iris bombé. It is then usually done by passing a Graefe knife across the anterior chamber, puncturing and counter-puncturing both cornea and iris. The iridectomy is performed before the punctures become closed with exudate, which usually occurs a short time after the operation.

Division of Anterior Synechiæ is a form of iridotomy.

It is most easily done by the use of Lang's twin knives, two tiny but similar knives, one with a sharp point and blunt edges, the other with a blunt point and a sharp edge. The first is introduced through the cornea near the limbus some distance from the synechia at a point which provides a good fulcrum and then withdrawn. The second is introduced through the same puncture, with the sharp edge facing the synechia. These manœuvres should be done without losing aqueous.

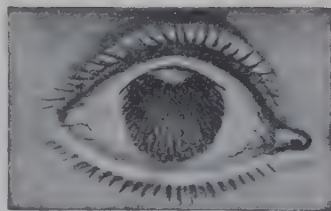


FIG. 342. Drawing up of the iris after extraction of cataract with incarceration of the pillars of the coloboma.

The second knife is then swept across the synechia cutting it close to the cornea and then withdrawn. If the synechia is extensive, two or even more operations may be required, a portion of the adherent iris being severed each time.

OPERATIONS FOR SIMPLE GLAUCOMA

Sclerotomy. If puncture of the posterior segment of the globe is required it is usually most safely performed through the pars plana of the ciliary body, 8 to 9 mm. behind the limbus to avoid the risk of a retinal detachment.

Anterior Sclerectomy is the name given to various operations for simple glaucoma in which a piece of sclera is excised to form a filtering scar. It will be convenient to note all the operations for

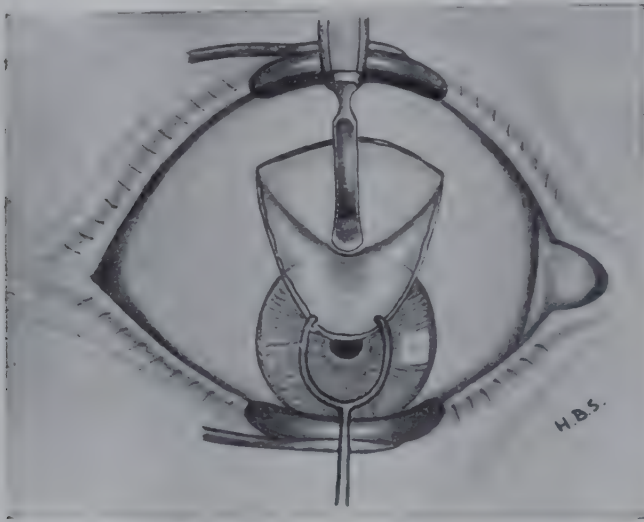


FIG. 343.

simple glaucoma at this point, although strictly speaking they are not all sclerectomies.

In *Elliot's trephining* the conjunctiva is seized with fixation forceps 8 or 9 mm. above the cornea. A large flap of conjunctiva and Tenon's capsule is made, almost concentric with the margin of the cornea (Fig. 343): the ends of the wound should be well away from the limbus, otherwise filtration is likely to be impeded owing to cicatricial tissue. The flap is dissected down to the upper part of the corneal margin and turned down over the cornea, being held there by forceps or a hook. The dissection is made by sharp-pointed scissors in the subconjunctival tissue until the edge of the cornea is clearly defined, care being taken to avoid button-holing the flap. The dissection is carried into the cornea (Fig. 343) so that the superficial lamellæ are dissected up with the flap for about 1 mm. with a Tooke's corneal splitter (Fig. 327). The trephine (1.5 or 2 mm. diam.) (Fig. 329) is then applied, so that half the aperture lies on the cornea, the other half on the sclera. The corneo-scleral

disc is cut by a few rotatory movements. When the anterior chamber is entered aqueous escapes, and the pupil is displaced upwards. The trephine is removed, a knuckle of iris protrudes from the wound and the disc is forced out; it usually remains attached by a small hinge. By tilting the trephine slightly forwards so that the corneal side of the disc is cut rather more deeply than the scleral, it is generally possible to ensure that the hinge shall be on the scleral side. The root of the iris is picked up with iris forceps, drawn slightly outwards and a small piece excised to form a peripheral iridectomy (Fig. 344). The disc is then seized with Elliot's disc forceps (Fig. 307) and excised with spring scissors (Fig. 311). The cornea is gently stroked downwards with a repositor until the pupil is round and the iris clear of the trephine hole. During the manœuvres the assistant must keep the flap well stretched downwards so as to avoid it also being button-holed—a rather serious accident. The flap is then replaced and fixed by a continuous

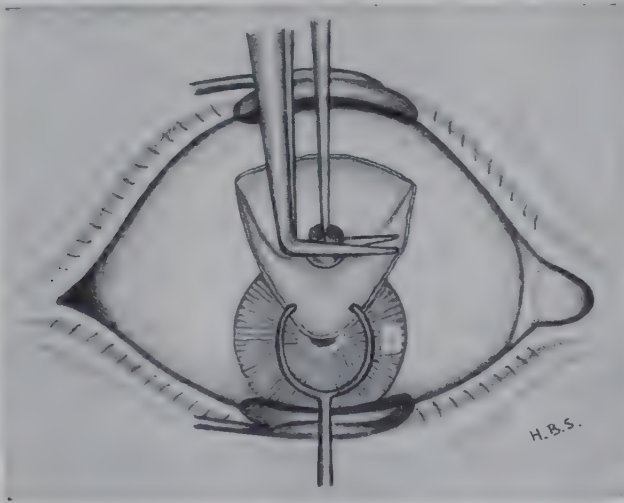


FIG. 344.

suture preferably in two layers, one of Tenon's capsule and the other of conjunctiva.

There is some tendency for iritis to develop immediately after trephining. Hence atropine and topical steroids should always be used daily for at least a week.

The chief *complications at the operation* are making the trephine hole too peripheral, often with consequent escape of vitreous and incarceration of the ciliary processes; button-holing the flap either with the trephine or de Wecker's scissors; escape of the corneo-scleral disc into the anterior chamber; and non-presentation of iris in the wound. The last complication is usually due to slow escape of aqueous owing to the trephine being blunt.

Post-operative complications include delay in the re-formation of the anterior chamber. In rare cases it never re-forms and the lens becomes opaque. Later complications are iritis, detachment of the choroid (p. 258), blockage of the wound with iris, ciliary body, lens or vitreous, or failure of filtration from dense cicatrization.

Owing to the prominence and thinness of the overlying conjunctiva *late infection* may occur long after the operation—a rare but serious event.

If trephining fails it can be repeated at some other part of the limbus, but the wound should always, if possible, be placed where it is covered and supported by the lid, although it should not be too close to the previous site.

Other filtration operations, some of them of great value, have been devised. In *Lagrange's operation* an ordinary iridectomy is performed, but before closing the wound a small piece of its anterior lip is snipped off without wounding the conjunctival flap. In *Scheie's operation* of cauterization of the sclera with a peripheral iridectomy, more permanent drainage is usually ensured. The sclera is lightly cauterized along a line 1 mm. behind the limbus underneath a reflected conjunctival flap, an incision is made with a knife along this line, the posterior lip of the wound thus made is again cauterized, the incision is completed to enter the anterior chamber, a peripheral iridectomy is performed and finally the conjunctival flap is sutured in place.

In *iridencleisis* a tongue of iris is incarcerated in a scleral incision near the limbus and partially severed from the main body of the iris to prevent the progressive bulging which tends to follow an iris prolapse.

A thick conjunctival flap is turned down to the limbus as in the operation for trephining, and an angled keratome (Fig. 317) pushed into the globe parallel to the plane of the iris from a point 1 mm. on the scleral side of the limbus, making an incision some 4 mm. long. The iris is grasped by iris forceps near the pupillary margin at two points on either side of the vertical meridian and cut from pupillary border to root. The two separated tongues of iris are torn apart for a few millimetres at the root, each is engaged in opposite sides of the scleral incision, and the conjunctival flap sutured in position over them.

In *cyclodialysis*, which is useful in aphakic eyes, a channel is opened up between the anterior chamber and the supra-choroidal space. Under a conjunctival flap an incision about 3 mm. long is made in the sclera 4 mm. behind and concentric with the corneo-scleral junction in the lower temporal quadrant. A spatula specially curved to fit the inner aspect of the sclera is inserted and passed forwards between the scleral spur and the ciliary body into the filtration angle, the point being tilted towards the sclera during the manoeuvre (Fig. 332). Here it is swept transversely through a small arc, breaking down the trabeculae and adhesions between the root of the iris and the cornea. The conjunctival flap is sutured over the wound.

Partial destruction of the ciliary body is a further method of dealing with a recalcitrant case of glaucoma. This may be done with diathermy (*cyclodiathermy*) whereby a partially penetrating or a surface

electrode is used to make a ring of applications 3 to 6 mm. behind the sclera. The entire ciliary body may be induced to undergo some degree of atrophy in this way, but an operation involving the whole ciliary body is best done in two or three stages. The first type of electrode is more safely employed under a conjunctival flap; the second can be applied to the conjunctiva. An alternative procedure is to induce ciliary atrophy by *cryosurgery* through the conjunctiva (p. 413).

Trabeculotomy. See p. 289.

OPERATIONS FOR CATARACT

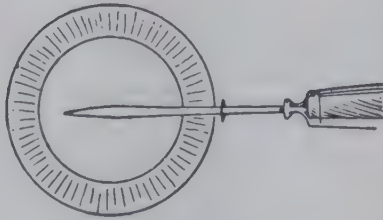
Discission or Needling of the intact lens is the most suitable operation for cataract before the age of fifteen; it may be employed up to the age of thirty, but the nucleus of the lens is then likely to give trouble. Discission is used at any age for the division of dense secondary cataract (after-cataract).

Needling of the soft lens in young patients usually requires a general anæsthetic; the pupil must be fully dilated with atropine.

It is best to perform the operation in a darkened room with oblique illumination.

The eye is fixed with fixation forceps and the discission needle is introduced just outside the limbus through the conjunctiva and sclero-cornea in a plane parallel to that of the iris, at a point just above the horizontal meridian of the cornea (Figs. 324, 345). It is carried through the anterior chamber until the point reaches the lower part of the pupil.

FIG. 345. Diagram of discission with one needle.



The handle is then slightly raised, so that the point perforates the lens capsule. The handle is then moved so that it and the point move through arcs of circles which have their centre at the spot where the shaft is engaged in the corneo-sclera. Having thus made a curved, more or less vertical incision in the capsule and the anterior cortex, a second incision is made at right angles to it. This is done by very slightly withdrawing the needle so as to disengage it, passing it farther on towards the far side of the pupil and bringing the cutting edge in contact with the capsule. As the needle is slowly withdrawn a straight horizontal incision is made in the capsule. When this is sufficiently large the handle is depressed and rotated so that the plane of the blade faces upwards, and the needle is quickly withdrawn from the eye. By withdrawing it quickly no aqueous should be lost, otherwise an anterior

synechia may result. Sterile atropine ointment is introduced into the conjunctival sac, and the eye is bandaged.

The most important point about after-treatment is keeping the pupil well dilated, which is done by atropine ointment two or three times a day. There is always some ciliary reaction. The amount of swelling of the lens fibres depends upon the size of the incisions in the capsule, but also varies with different lenses. If it is desired to avoid the necessity of a subsequent curette evacuation, the incisions should be small but a second or third needling at intervals of several weeks or even months will usually be necessary before the lens substance is absorbed. If it is intended to complete the procedure in one operation, the incisions may be as large as possible, and the needle may even be introduced deeply into the lens and the fibres broken up; the lens substance may then be aspirated out through a syringe or a tube. Alternatively, the eye may be left for some days; in this case there is swelling of the lens and the anterior chamber becomes filled with flocculent masses while there is intense ciliary injection accompanied by raised tension and perhaps pain. Thereupon a curette evacuation should be performed (p. 425). In all cases a final needling will be of the type of a discission for secondary cataract.

Discission of secondary cataract (after-cataract) (capsulotomy) may be performed in exactly the same manner as discission of the lens.

A curved Ziegler's knife-needle (Fig. 323) is probably the best instrument. This is inserted near the limbus with the blade flat, and when the iris has been passed, the point of the knife is threaded under

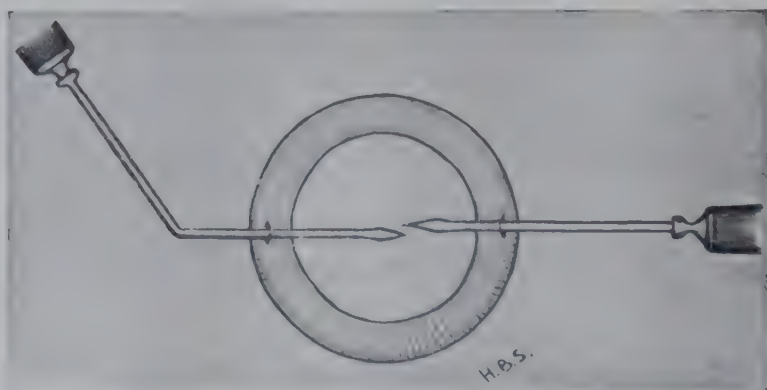


FIG. 346. Diagram of discission with two needles.

the capsule and brought out again at the opposite side of the pupil and cut outwards through the capsule. In this way trauma to the face of the vitreous is minimized. If the capsule is dense and thickened, two needles may be employed, the shaft of the one used on the nasal side being bent to an angle of 135° (Fig. 346). By this method, while the eye is fixed by an assistant, the capsule can be steadied by the first needle

while the other is used for cutting so that no undue strain is thrown upon the ciliary body, and the membrane is prevented from tearing away from the ciliary body instead of being torn in the centre.

Needling operations are by no means so simple as they appear in the hands of an experienced operator. Every movement of the needle must be made round the spot where the shaft penetrates the globe. Moreover, no undue traction should be put on the capsule lest the trauma thus set up induce an immediate post-traumatic iridocyclitis or a delayed detachment of the retina. Indeed, when the membrane is very dense it is often best to divide it with scissors, as in iridotomy (p. 419).

Curette evacuation or linear extraction is the operation whereby after discission, whether accidental (traumatic cataract) or intentional, the softened lens matter is let out of the anterior chamber.

The pupil must be fully dilated with atropine. A keratome (Fig. 316) is passed through the cornea, 1 mm. internal to the apparent margin (*cf.* wound in Fig. 338), with the blade parallel to the plane of the iris, and pushed on until the incision is about 5 mm. long. The point may be dipped so as to pass into the lens without disadvantage, and the incision may be increased as the keratome is being slowly withdrawn by extending each angle, using the two edges of the keratome like knives. A piece of capsule may be removed by capsule forceps (Fig. 308) thus diminishing the risks of dense after-cataract in the pupillary area. The tip of the curette (Fig. 321) is then gently insinuated just within the edges of the wound. Slight pressure is exerted upon the scleral lip, and the soft lens matter travels along the groove of the curette. No attempt should be made to remove the whole of the lens matter on account of the danger of rupture of the suspensory ligament and the escape of vitreous, but as much as possible should be washed out of the anterior chamber as in the operation for extracapsular extraction (p. 430); the remnants will be absorbed. There is no probability of the iris prolapsing or becoming incarcerated in the wound if it is properly dilated, but the repositor should be gently inserted so as to push back any lens capsule which may be prolapsed.

Extraction of senile cataract may be performed by several methods. In *extracapsular extraction* the capsule is opened, as much of the lens as is possible expressed and the capsule left behind so that a subsequent needling is required (p. 423). In *intracapsular extraction* the lens is removed in its capsule and only one surgical intervention is required. It is traditional to designate an extracapsular operation which includes a complete iridectomy a *combined extraction*, that in which the iridectomy is omitted or peripheral, a *simple extraction*.

All cataract operations should be preceded by an unusually careful overhaul of the patient and an examination of the conjunctival sac and lacrimal sac. Full mydriasis, regional analgesia, akinesia and control of the eye by a superior rectus stitch (p. 408) are necessary, and a speculum which does not press upon the globe should be used or, alternatively, lid stitches.

In the various types of operation two common factors will be discussed first—the section and the use of sutures.

The *section* can be made in the classical way with the Graefe knife (Fig. 314) or with the keratome (Fig. 316) and corneal scissors (Fig. 311). A good section is probably the most important single factor in determining the success of the operation.

For the *Graefe section* the surgeon stands above the head of the patient, making the section with his right hand for the right eye, and with his left hand for the left eye. Some surgeons stand below and at the side for the left eye, and cut away from themselves, using the right hand. The eye is fixed with fixation forceps applied over the lower border of the insertion of the medial rectus muscle. The point of the Graefe knife is inserted in the apparent corneo-scleral margin at a point 1 mm. above the horizontal meridian of the cornea, care being taken that the cutting edge is upwards (Figs. 347, 348). As soon as the point is in the anterior

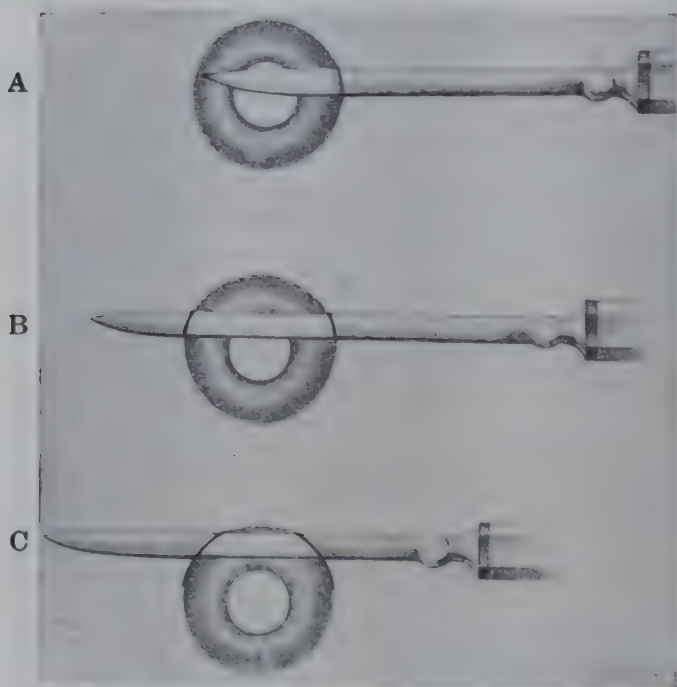


FIG. 347. Cataract section. A. Commencing the counter-puncture. B. Counter-puncture made. C. Commencing the section through the limbus.

chamber it will look much brighter than the part in the cornea; if this is not noticeable it is probable that the knife is badly directed and has burrowed in the cornea. It is passed steadily onwards across the anterior chamber to the corresponding spot on the opposite side, the point being aimed to emerge 1 mm. on the corneal side of the limbus. Owing to its apparent displacement forwards by the corneal refraction, it will in this event emerge at the corneo-scleral margin. The knife is made to cut out by a series of small sawing movements, little pressure upwards being

required with the very sharp knife. It is very necessary to use these sawing movements properly, as the sharpest knife fails to cut if it is simply pressed hard against a surface. A conjunctival flap has already been cut at the sides, but the middle of the knife blade is still under the conjunctiva. The edge of the knife is then directed forwards and the conjunctiva cut through by one or two sawing movements.



FIG. 348. Diagram of wound in extraction of senile cataract.

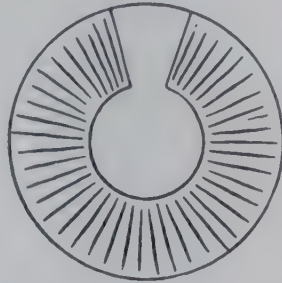


FIG. 349. Diagram of coloboma in cataract extraction with iridectomy.

The size of the section depends upon the probable size of the nucleus of the lens. In senile sclerosis the whole lens is hard, so that a large section must be made involving half the circumference of the cornea. More harm is done by bruising the edge of the wound than by having a wound which is unnecessarily large. In making a Graefe section the aqueous may escape prematurely, so that the iris floats up in front of the knife. If this happens the knife should be raised as if to lift the eye forward. If this manœuvre fails, the section must be completed in the usual manner, although the iris is wounded, either a hole or a complete coloboma being cut out of it. Cutting the iris causes pain unless regional anæsthesia has been given and is likely to make the patient flinch, screw up the eye, or completely lose self-control. Moreover, the coloboma is generally irregular. The accident is usually due to hesitation in pushing the Graefe knife steadily forward or to pressure—often unconscious—exerted on the eye by the fixation forceps. It is least likely to occur if the passage of the knife through the anterior chamber and the commencement of the section are all part of a single steady forward sweep of the blade, the handle of the knife being depressed directly the counter-puncture is made. In this manner the broad part of the blade is brought over the iris as quickly as possible. There should, however, be no haste, every movement being made with deliberation but not too slowly.

In the *keratome section* a conjunctival flap 3 mm. in breadth is dissected down to the limbus with scissors around the upper half of the eye above the horizontal meridian and its upper part reflected over the cornea. While the eye is steadied by the superior rectus stitch, a straight keratome (Fig. 316) is then inserted at the upper pole of the limbus and pushed steadily into the anterior chamber in the plane of the iris, its sides cutting a limbal incision from about 11 to 1 o'clock. At each end of the incision one blade of the spring scissors (Fig. 311) is

then carefully inserted into the anterior chamber running round the angle between the iris and the cornea, the other underneath the conjunctival flap, so that, on closing the scissors, the incision is prolonged to a short distance above the horizontal meridian. This manoeuvre should be accomplished in a single snip on each side. To the unskilled this technique provides great accuracy in the location of the section, a matter often of importance when preplaced sutures are employed, and ensures the protection of a broad and complete conjunctival flap.

Sutures should always be used in cataract surgery; they ensure rapid and accurate apposition of the wound, lessen the likelihood of prolapse of the iris, allow the unoperated eye to remain uncovered, and shorten the period of post-operative recumbency and enforced quietude. With efficient corneo-scleral suturing the patient can sit up in bed the day following operation and get up out of bed on the next day if necessary or on the third or fourth day if desirable—an important consideration in old people, particularly with chest complications.

A multitude of suturing techniques has been devised, many of them unnecessarily elaborate. One or at most two corneo-scleral sutures only are necessary, while the conjunctival flap can be secured after the operation by a few conjunctival sutures. The corneo-scleral sutures are most safely inserted before the section is completed; they are left slack and tied after the lens is removed; if silk sutures are employed they are removed in eight or nine days' time; this procedure, which is always painful and sometimes difficult, may be avoided by the use of fine catgut sutures. Fine and very sharp atraumatic corneo-scleral needles are essential. The sutures should never penetrate the whole thickness of the cornea lest an ingrowth of epithelium occur into the anterior chamber along their tracks, and they should never be tied more tightly than is required to coapt the edges of the wound lest the compressed tissue necrose. The most accurate apposition is obtained by direct sutures passing through the outer half of the actual section, a technique which can be applied to the keratome incision before it is completed by scissors. The simplest is that illustrated in Fig. 350.

The needle is passed through the superficial layers of the cornea transversely for 2.5 mm. at a point about 1.5 mm. on the corneal side of the limbus; it is then passed through conjunctiva and sclera 2.5 mm. above and exactly opposite and parallel to the direction of the corneal suture (Fig. 350). The corneo-scleral section is made between the corneal and scleral insertions.

The *injection of air into the anterior chamber* through a fine-angled needle insinuated under the conjunctival flap at the end of the operation after the sutures have been tied, has some advantages. The chamber is re-formed immediately, the iris and vitreous pushed back to their original positions and the danger of prolapse of the iris lessened. In this event, however, a large bubble should completely fill the anterior chamber; a small bubble may insinuate itself behind the iris and make matters worse. The air is gradually absorbed and replaced by aqueous in two or three days.

Extracapsular Extraction: Combined Extraction. After the corneo-scleral sutures have been inserted and the section made, the patient is asked to look down towards his feet, the eye being controlled by the superior rectus stitch. The iridectomy is then performed.

The iris forceps are taken in the left hand and the de Wecker's scissors in the right. The forceps are inserted at the centre of the

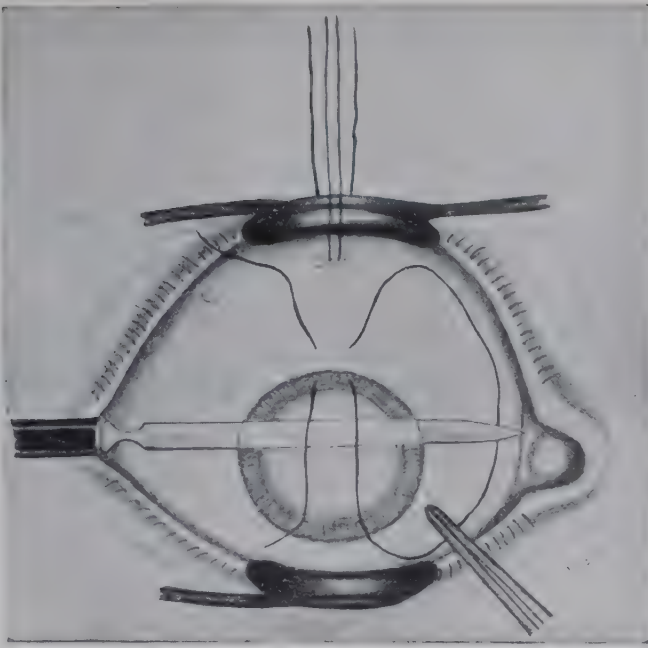


FIG. 350.

wound, passed to just above the pupillary margin of the iris and the iris seized and drawn out of the wound. The piece of iris grasped is cut off with one snip of the de Wecker's scissors, the blades of which are held radially to the iris (Fig. 340). The iris reposer is now taken and the iris replaced as in the operation for iridectomy (*q.v.*), particular care being taken to free it from the angles of the wound (Fig. 349).

The patient still looking towards his feet, the cystitome (Fig. 320) is introduced, the blade flat and the blunt curved edge leading, until the point is near the lowest part of the pupillary margin. The cutting

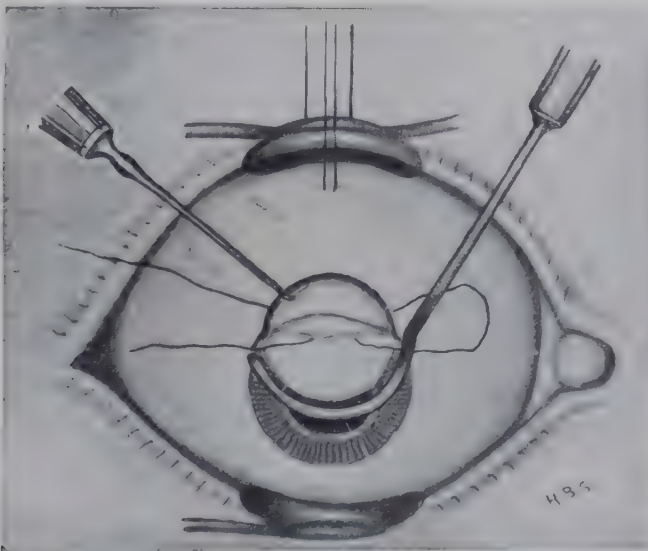


FIG. 351.

edge is then directed backwards, the handle is slightly raised, and the lens capsule is incised vertically and horizontally.

The cystitome is then taken in one hand, and the curette or the lens expressor (Fig. 325) in the hand corresponding to the eye being operated upon. The back of the curette or the lens expressor is placed horizontally upon the lower part of the limbus and gentle but firm pressure is made in a direction backwards and slightly upwards. This causes the nucleus of the lens to be tilted so that the upper edge appears presenting in the wound. The lens nucleus is coaxed out of the wound by continuing the pressure with the curette, but more and more in an upward direction. Meanwhile the lens may be gently helped out by the cystitome in the other hand (Fig. 351). As soon as the diameter of the nucleus has passed through the wound, pressure upon the cornea is relaxed. If there is much clear soft lens matter, this is coaxed out of the wound by irrigation of the anterior chamber with normal *sterile* saline at body heat through a cannula inserted into the anterior chamber and attached by tubing to an undine, held slightly above the level of the eye, which serves as a reservoir (Fig. 352). A flow unimpeded by air-bubbles should first be secured and the height

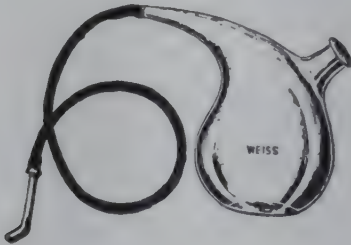


FIG. 352. Undine fitted with cannula for washing out the anterior chamber.

of the undine should not be too great so that a gentle stream is used. As much cortical matter should be removed as possible since its presence frequently gives rise to a post-operative iritis and makes subsequent dissection difficult.

The iris repositor (Fig. 331) is now again used to free the iris from the angles of the wound and push back into the anterior chamber any tags of capsule which may be presenting. These are so transparent as to be invisible, and it is extremely important that they should not become incarcerated in the wound. The corneo-scleral suture should then be tied, the conjunctival flap sutured to its normal position, atropine instilled and appropriate dressing applied.

Simple extraction is performed in the same manner as that already described except that the total iridectomy is omitted. In its place one or two small *peripheral iridectomies* should be performed as a precautionary measure to allow communication between the posterior and anterior chambers and thus avoid a possible subsequent prolapse of the iris into the wound. In this manœuvre the periphery of the iris is gently drawn out of the wound by iris forceps and the smallest possible fold is snipped off with de Wecker's scissors. The lens is expelled as already indicated and an iris repositor is inserted and the iris stroked back into position. The pupil should be quite round: if not, or if the iris tends to prolapse, an iridectomy is necessary. Thereafter the iris is again replaced by a repositor and the corneo-scleral suture tied.

Intracapsular Extraction. This is the most satisfactory procedure. After the section is made, a complete or small peripheral iridectomy is performed, according to the judgment of the surgeon. Capsule forceps (Fig. 308) are introduced, closed, to the temporal side of the iridectomy. On reaching the pupillary margin the blades of the forceps are moved over the anterior capsule in the sagittal plane and are stopped over the thickest part of the capsule just in front of the equator near the lower edge of the lens. The blades are then opened 2 mm., pressed gently backwards to engage the capsule, and closed. Gentle rotatory, zigzag movements are made so as to rupture the suspensory ligament (Fig. 353). These movements increase in excursion, and not until the lower edge of the lens is felt to be free and to move forwards is any attempt made to lift the lens and tumble it forwards. The remainder of the manoeuvre consists in holding the capsule without pulling on it, and using the lens expressor in the usual way, keeping it just below the lowest part of the lens during delivery (Fig. 354). When the lens is in the wound it is important to complete the final stage slowly and deliberately, allowing the vitreous

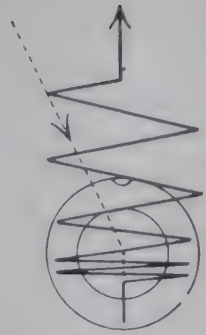


FIG. 353. Zig-zag movements made with the capsule forceps to rupture the zonule in intracapsular extraction. The dotted line indicates the line of introduction of the forceps (Arruga).



FIG. 354. Intracapsular extraction. On the left, the lens is being delivered by the capsule forceps assisted by pressure outside the cornea by an Arruga's expressor. On the right, the manoeuvre seen in section (Stallard).

to gravitate and the iris to slide back in place. When the lens has been removed the corneo-scleral suture is tied, a sterile drop of pilocarpine (2 per cent.) is instilled, the iris replaced, and the conjunctival flap adjusted and sutured.

As an alternative to extracting the lens with capsule forceps, cryosurgery is a very useful technique. The cryoprobe is placed upon the anterior capsule, care being taken not to touch the iris, whereupon the neighbouring cortex is frozen onto it allowing its delivery from the eye.

The difficulty in intracapsular extraction lies in toughness of the zonule, a condition most common in young and middle-aged adults. If the capsule is torn in the manipulations an extracapsular operation may be done, but this accident can be anticipated by the use of *alpha-chymotrypsin* (*zonulysin*), a proteolytic enzyme which dissolves the zonule. A 1 in 5,000 solution is injected through the corneal section into the posterior chamber and after 2 or 3 minutes is thoroughly irrigated out; this is essential lest the enzyme damage other ocular tissues including the corneal endothelium. Thereafter the partially digested zonule often gives way so readily that the lens may well be extracted by simple external expression. The method should not be used in children in whom the vitreous may be adherent to the posterior capsule of the lens.

Operative Complications. Whichever type of operation is employed, complications may occur. If the nucleus does not come forward through the wound with moderate pressure of the curette, the fault is probably that the section is too small. The wound should then be enlarged with scissors (p. 427). Occasionally in extracapsular extractions the difficulty is due to the capsulotomy having been inefficiently performed; this is overcome by more careful repetition of the capsulotomy.

If the sclera is rigid the cornea collapses and becomes saucer-shaped. This is of no consequence and requires no action, nor does any bubble of air which may enter the anterior chamber; it soon becomes absorbed.

Hæmorrhage may occur into the anterior chamber. The blood is derived from the wound or from a hyperæmic iris. An attempt may be made to wash it out by irrigation before it clots, or to remove the clot with forceps.

The most serious accident which may occur during extraction is prolapse of vitreous at an early stage. It may be due to inherent weakness of the suspensory ligament, which gives way during the section or to clumsy technique such as undue pressure on the eye by the fixation forceps or with the curette in the attempt to expel the lens.

If the vitreous wells into the wound spontaneously, or on attempts to expel the lens without any sign of the engagement of the edge of the lens in the wound, it is a good plan to discard a speculum and rely on lid stitches to keep the eye open and to resort at once to scoop extraction. It is thus important to have the scoop or vectis always in readiness in every case of extraction (Fig. 322).

In *scoop extraction* the spoon is passed directly backwards into the vitreous so as to make certain that it passes behind the lens. It is then rotated forwards so that the lens is caught between the spoon and the back of the cornea. The lens is kept pressed against the cornea and is removed by a rapid further rotation of the spoon. Some vitreous may be lost, but it is imperative that the lens should be delivered if the eye is to be retained. The eye in these cases is closed with as little toilet as possible since any further manœuvre is likely to lead to greater loss of vitreous. In other cases badly managed, the lens may sink back into the vitreous. In this event it is usually futile to attempt to remove it. The eye should be dressed at once, and if the lens floats

up into the pupillary area at a later date a further attempt may be made to extract it with the scoop.

Prolapse of the vitreous after delivery of the lens is less serious, although it increases the tendency to cyclitis with opacities in the media, and may be followed by a detachment of the retina. If much vitreous is lost the iris is usually gradually drawn upwards, so that in course of weeks or months the pupil is displaced and the lower part of the iris stretched (Fig. 342). This condition may also occur from incarceration of the pillars of the coloboma in the wound. It may be necessary to do an iridotomy or some such operation to make an artificial pupil so that vision may be restored (p. 419).

After-treatment. The patient lies quietly upon his back with his head and shoulders raised, in the semi-sitting (Fowler's) position, and is directed to avoid all straining. A sneeze may be inhibited by pressure with the finger on the upper lip close to the septum of the nose. The slight amount of post-operative aching is readily controlled by sedatives. Unless day-and-night nursing is available it is a wise precaution with many patients to tie their hands loosely to the bed at night lest they knock or rub their eyes when they are half asleep. The food must be soft during the first few days ; no aperient is given for two days.

On the day following the operation the eye is dressed using swabs with warm lotion and, in an extracapsular extraction, a drop of sterile 1 per cent. atropine solution instilled ; this is not necessary in an intracapsular extraction in which the eye remains white. The wound is inspected, but should not be disturbed more than is absolutely necessary.

On the second day the cornea should be bright, and the pupil round. Faint greyness in the cornea above (striate opacity, p. 221) need cause no alarm. In an extracapsular extraction atropine is instilled and continued for a week ; this should be done in any type of extraction if there are signs of iritis, supplemented if necessary after three days by a corticosteroid ointment. The eye should be almost free from injection in a week or ten days.

It is wise to keep healthy patients in bed for four days with the operated eye covered. Exception, however, may well be made for old and debilitated people who should be propped in bed immediately after operation and it may be advisable to allow them to get out of bed on the second or third day. If they show any signs of early mental disturbance or wandering in speech and the unoperated eye is blind, the operated eye should be uncovered and dark glasses substituted ; this is relatively safe in the presence of efficient suturing. Normally, however, a light dressing is kept on for a week ; afterwards tinted glasses are worn ; temporary cataract glasses can be ordered after two and permanent spectacles in about six weeks.

The *post-operative complications* of cataract extraction are striate keratopathy (p. 221), incarceration of the iris in the angles of the wound,

prolapse of the iris, iridocyclitis, sympathetic ophthalmitis, secondary glaucoma, intra-ocular hæmorrhage, infection of the wound and panophthalmitis.

Prolapse of the iris is most effectively prevented by a peripheral or total iridectomy, the use of sutures and the injection of air into the anterior chamber. It usually occurs in the first day or two, but may result later from injury to the eye by rubbing or knocking it, straining or coughing. It must be treated *at once* by abscission of the prolapse. As the iris is irritable the operation is painful, and general anæsthesia or retrobulbar local anæsthesia is generally necessary. The wound is reopened by insinuating the tip of an iris repositor under the conjunctival flap and gently uncovering the prolapse. The flap is turned down over the cornea, the iris pulled out with iris forceps and snipped off with de Wecker's scissors. The iris is then replaced with a clean repositor and the conjunctival flap brought back into position. A small subconjunctival knuckle of iris can sometimes be replaced by an iris repositor, but it is generally wiser to snip it off.

Delayed Re-formation of the Anterior Chamber may be due to a jagged section, over-riding of the lower lip of the wound, or to no apparent cause. It is much less common in cataract extraction than after iridectomy or trephining for glaucoma. In these cases the bandage should be very lightly applied or discarded, dark protective goggles being worn in the daytime, and a light bandage with a wire or cartella shield at night. The administration of Diamox may be helpful and the injection of air into the anterior chamber may be tried.

Expulsive Hæmorrhage is fortunately rare. It occurs during or soon after operation in old people with arteriosclerosis. There is sudden severe pain, and on removal of the dressings the wound is found to be gaping and filled with blood clot, vitreous, etc. The eye is always lost and should be excised. This may be necessary in order to stop the bleeding, the socket being then packed and firmly bandaged.

Septic Infection may occasionally occur, especially in diabetic patients, from the twelfth to thirty-sixth hour after operation. There is severe aching pain, due to the accompanying acute iridocyclitis. On removing the dressings the upper lid is œdematous. When the lids are separated tears gush out and there is muco-pus in the conjunctival sac. The lids should be separated gently, if necessary with retractors. The cornea is then seen to be dull and hazy, especially in the upper part, the lip of the wound being yellow, intense iritis is present, the pupil and coloboma become filled with exudate, a hypopyon appears and, finally, the vitreous becomes infected and panophthalmitis leads to the destruction of the eye. Treatment as for panophthalmitis (p. 245) should be applied quickly and energetically but is seldom effective.

Iritis in mild degree probably occurs in all cases of extracapsular cataract extraction. In its more pronounced form it is especially associated with retained lens matter (p. 430) and diseases such as diabetes, rheumatism or gout. The worst cases occur with acute septic infection. Intermediate in severity are cases of plastic iritis due to infection by less virulent organisms or in patients with greater resistance to bacterial invasion. More insidious than any are cases of continued irritability of the eye with mild iritis and keratic precipitates. Both in these and in cases of plastic iritis there is danger that the

condition is really sympathetic ophthalmitis. Hence it is essential in all cases of cataract extraction to inspect the cornea most carefully with oblique illumination and the loupe and to do so frequently, especially if there is an unusual degree of flushing and lacrimation on exposure to light. The other eye must also be carefully watched. If there are "k.p." no needling operation must be undertaken until the eye has quietened down and remained quiet for many weeks. It is sometimes difficult to distinguish minute spots of lens substance on the back of the cornea from true "k.p."; the former soon become absorbed. In all such cases treatment by atropine, antibiotics and steroids, topically and if necessary systemically, should be intensive.

Detachment of the Choroid. See p. 258.

Secondary Glaucoma may occur after cataract extraction, usually due to pupillary blockage, peripheral anterior synechiæ or incarceration of the capsule in the wound. Sometimes it is due to the anterior chamber being lined with epithelium when there has been delay in healing and the conjunctival epithelium has grown down into the anterior chamber and spread over the surface of the iris, lens capsule and cornea. These cases are difficult to diagnose and to treat. Sometimes secondary glaucoma follows needling of an after-cataract; it is usually then attributed to vitreous extending into the anterior chamber and interfering with filtration, but it is doubtful if this is the invariable explanation. These cases often do badly. Where there are definite adhesions of the capsule an attempt may be made to divide them; ingrowing epithelium is best treated by irradiation; in the more obscure cases a cyclodialysis is usually the operation of choice, and if it fails a cyclo-diathermy may be tried.

OPERATIONS FOR RETINAL DETACHMENT

Operations for retinal detachment can only be performed after accurate localization of the retinal hole (p. 335). Anæsthetic drops should not be used to attain analgesia since they cloud the cornea and preclude accurate ophthalmoscopic examination. Analgesia is obtained by subconjunctival, sub-Tenon or retrobulbar injections, the first being introduced relatively painlessly through the skin of the lower lid (Fig. 355).

In the operation using *diathermy* a flap of conjunctiva and Tenon's capsule is reflected over the site of the retinal hole. Owing to the fact that the terminal of the diathermy can, if required, be slipped under an extra-ocular muscle, it is rarely necessary to reflect these unless a posterior position of the retinal hole makes it necessary to rotate the eye to an extreme degree to gain access to the site. An application of surface diathermy is then made at the presumptive site of the retinal lesion and the intensity and location of the reaction checked by ophthalmoscopic examination. Its relationship to the retinal lesion determines the sites of further applications of surface diathermy. After satisfactory circumvallation of the hole or holes by surface diathermy as checked by ophthalmoscopic examination, one or more perforations of the sclera and choroid are made by the needle-terminal or a fine cautery through which the subretinal fluid seeps out. Ophthalmoscopic examination should then show apposition of the retina to the choroid; if it is not so apposed, air or sterile vitreous from a cadaver eye may be introduced

into the vitreous through a separate perforation in the region of the ora to force it against the choroid. Both eyes are bandaged, and the head is immobilized in a position such that the torn portion of the retina tends to fall gravitationally on the underlying choroid. In favourable cases adhesion between retina and choroid is fairly firm in two weeks, but for some time great care must be taken not to jeopardize its security by undue physical exertion.

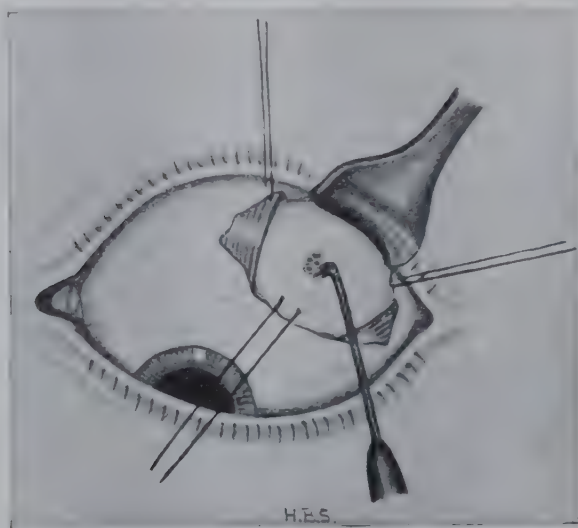


FIG. 355. Perforating diathermy through the site of superficial diathermy.

An alternative method is to induce a similar sterile choroiditis in the region of the retinal hole by *cryosurgery*; if the retinal hole is near the periphery this may be done through the intact conjunctiva.

A third technique is to cause a choroidal burn by *light-coagulation* in the region of the retinal hole. Since the light is absorbed by the pigmentary epithelium, this is only effective if the detachment is shallow.

Another approach may be made through scleral surgery when the retinal holes are numerous or large and the detachment has ballooned. In these cases the sclera may be made to approach the retina by a *lamellar scleral resection*. In this operation the superficial two-thirds of a strip of sclera over the retinal hole is dissected away, the remaining third is lightly cauterized and the edges of the wound apposed with interrupted sutures, thus turning the inner third of the sclera inwards towards the retina. Light-coagulation or cryosurgery may then be used, if necessary, to induce further adhesions between the retina and the choroid.

In more severe cases a *scleral buckling operation* may be undertaken wherein the sclera is indented over the retinal hole by stitching onto it an implant of silicone rubber. Alternatively, when there are many holes, an *encircling operation* may be tried; in it a band of silicone rubber is passed round the eye beneath the rectus muscles and tied after release of some subretinal fluid so as to produce a circumferential buckle. In these cases also further adhesions between the retina and the choroid can be ensured by light-coagulation or cryosurgery.

EXCISION OF THE EYEBALL

The appalling tragedy of removing the wrong eye is known to have occurred. The accident is most likely to happen when there are no superficial evidences of disease, as for example in excision for a tumour of the choroid. It is well to mark the forehead above the eye to be removed before the anæsthetic is administered, or to re-examine the fundus immediately before surgery.

A general anæsthetic is usually given, but if desired the operation can be performed almost painlessly under regional analgesia. The technique is as follows :—

After the speculum is inserted, the surgeon seizes the conjunctiva just outside the limbus at the upper part of the cornea with fixation forceps and incises it here with blunt-pointed scissors (Fig. 312), one blade being passed under the conjunctiva as far as possible round the cornea. By carrying the point out under the looser bulbar conjunctiva it may be taken a third of the distance round the circumference ; the edge is then brought close up to the limbus before the conjunctiva is divided. The manoeuvre is repeated on the other side of the cornea. Finally, the portion below the cornea is divided. The conjunctiva should be divided completely round the cornea, and close to it, in three or four cuts. The peripheral edge of the cut conjunctiva is then taken up by the forceps, and the bulbar conjunctiva is separated from the globe as far back as the equator in all directions by a series of small snips, the blades of the scissors being kept flat in close contact with the eyeball. In this manner the capsule of Tenon is simultaneously opened.

The tenotomy hook (Fig. 326) is then taken in the left hand, the scissors being retained in the right. The rectus muscles are taken up one by one and divided close to the globe. It is well to begin with the superior rectus, since it is the most difficult to reach. The tendon of the lateral rectus of the right eye or the medial rectus of the left eye should be left long. The obliques are found by passing the hook farther back and carrying it round close to the globe.

The speculum is then taken and held widely open and pressed back into the orbit. If the muscles have been properly divided the globe springs forwards between the blades of the speculum. If it does not do so the speculum is removed and the eye dislocated by pushing the lids behind it with the fingers ; in children, in whom the orbit is small in relation to the size of the globe, it may be necessary to lever it out with the points of the excision scissors. The heavy excision scissors are taken in the right hand, the appropriate long tendon stump seized with toothed forceps and the globe pulled forward and rotated towards the left side of the patient so that the optic nerve is easily reached. The points of the closed scissors are passed into the orbit—to the outer side of the eye on the right side, to the inner on the left. The optic nerve is felt with the closed scissors : it is easily recognized. The scissors are withdrawn a short distance, opened, and the blades pushed down, one on each side of the nerve, which is then divided ; the sensation of dividing the nerve is unmistakable. The eyeball can then be freely drawn forwards so that any remnants of the obliques still attached

to the globe can be divided close to the eye. To control bleeding, the inside of the muscle cone is packed with gauze wrung out in hot saline and pressure kept up for two or three minutes. The edges of the conjunctiva are then drawn together by a continuous silk suture, antibiotic ointment inserted, the lids closed, and the dressing applied with firm pressure. This should consist of a small spherical pad of gauze, then a round flat pad of sterilized or cyanide gauze, then a thick round pad of sterilized wool. The bandage is applied also with firm pressure. The patient is kept in bed for one or two days. The suture is removed after forty-eight hours. Subsequently there is always some mucoid secretion which necessitates washing out the orbit twice a day with an astringent lotion.

If the globe is perforated and collapsed the rupture should be closed by sutures before proceeding to excise the eye.

It is easy to cut the sclera instead of the nerve, especially with curved excision scissors. Particularly when the nerve has to be cut long, as in excision for neoplasms, it is preferable to use straight scissors and to pass them along the inner wall of the orbit; indeed, straight instruments should always be used in preference to curved when possible, because it is easier to judge the position of the point.

Evisceration of the eyeball is recommended only in some cases of panophthalmitis (*q.v.*) in order to prevent the extension of the infection up the optic nerve sheaths.

In this operation an opening is made into the cornea which is then removed with scissors and all the intra-ocular contents are scooped out, the inner surface of the sclera being thoroughly cleansed with a swab. If the entire sclera is left there is considerable reaction and delayed healing. These disadvantages may be obviated by cutting the insertions of the extra-ocular muscles and excising the greater part of the sclera, leaving only a small collar of it around the optic nerve so as to leave the nerve sheaths unopened (*frill excision*).

Excision with the introduction of an implant has the advantage that it provides support for the artificial eye, making it look less sunken and endowing it with some mobility so that the simulation of a real eye is closer. All implants, however, unless firmly fixed and buried, have a tendency subsequently to be expelled.

The simplest technique is to bury a plastic implant in Tenon's capsule and to it the extra-ocular muscles are sutured. Cat-gut sutures are pre-placed in the muscle tendons before these are cut and the implant secured by suturing Tenon's capsule and the conjunctiva over it.

Implants have been used with an exposed surface provided with a hole into which a peg attached to the posterior surface of the artificial eye is inset. The immediate cosmetic result is good; but they are so frequently extruded subsequently that their use is not recommended.

An *artificial eye* of glass or plastic should not be worn less than 2-3 weeks after excision. A small eye may be worn for an hour or two a day until the conjunctiva becomes used to the foreign

body. Eight or nine weeks after the operation a full-sized eye may be worn; a plastic eye need be taken out and washed only once a week. Glass eyes tend eventually to become rough from chemical action of the secretion, in which event they must be discarded; a new glass eye should be obtained every year. Plastic eyes are more comfortable to wear than glass eyes and have the great advantages of being unbreakable and of adapting themselves readily to the temperature of the socket—a great advantage in intense cold; they are also more durable, but occasionally an idiosyncrasy to plastics occurs when irritation ensues. The methods of insertion and removal of the artificial eye should be learnt by every surgeon by seeing it done.

Contracted Socket is the result of injury, faulty excision, cellulitis in the orbital tissues, or the continued wearing of a rough artificial eye. The first three causes lead to the formation of dense cicatricial bands across the socket, rendering the wearing of a prosthesis impossible. The last cause usually results in obliteration of the lower fornix, so that the eye cannot be kept in place.

Contracted sockets are difficult to remedy. It is easy to divide the bands and make a new groove to hold the eye in position, but unless the wounds become covered with epithelium the edges heal together and no improvement is produced. A thorough dissection of all fibrous bands should be made, and the raw surface covered by a Stent mould. The Stent is cooled *in situ* by drops of cold saline and removed. A clamp is applied to the lower lip, which is then everted to expose its inner aspect. The Stent mould is placed over this, and a graft of buccal mucous tissue nearly twice the size is cut. The submucous tissue is dissected off and the graft sutured in position in the orbit. The Stent is secured in place over this by mattress sutures; the eyelids are closed and covered with a dressing of tulle-gras gauze wrung out in saline, and a pad and bandage are applied. This grafting procedure, however, is not by any means invariably successful.

In bad cases of contracted socket restoration can only be obtained by dissecting away all the remaining conjunctiva and fibrous tissue and grafting an ample split-skin graft with the aid of a Stent mould. In skin-grafted sockets, however, there is always an unpleasant discharge and smell from desquamating epithelium, and it is usually preferable in such cases to close the lids with a permanent tarsorrhaphy or wear an eyeshade, leaving the socket alone.

EXENTERATION OF THE ORBIT. See p. 528.

SECTION V

DISORDERS OF MOTILITY OF THE EYES

CHAPTER 28

ANATOMY AND PHYSIOLOGY OF THE MOTOR MECHANISM

IF each eye is to be rapidly and accurately fixed upon any object so that its image is thrown upon the fovea, and if the two eyes in their every movement are to move in unison so that binocular vision is to be attained, it is obvious that their motility and co-ordination must be subserved by an unusually accurate and responsive neuromuscular apparatus. We shall first study the extra-ocular muscles and then their central nervous control.

The Extra-ocular Muscles. A team of six muscles controls the movements of each eye. Four of them, the *rectus muscles*, have the



FIG. 356.

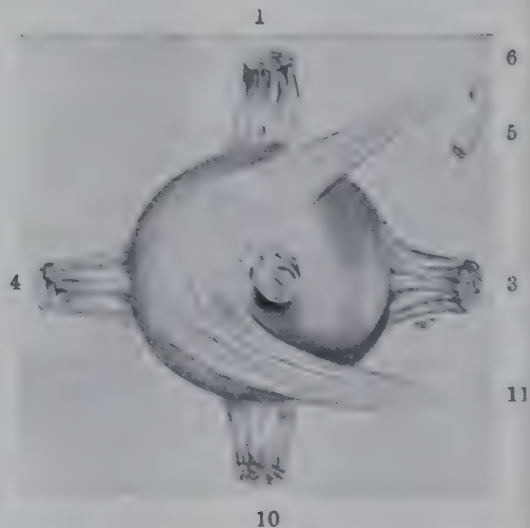


FIG. 357.

FIGS. 356-7. The extra-ocular muscles. Fig. 356, view from above (right eye); Fig. 357, view from behind (left eye). 1, superior rectus; 2, levator palpebrae superioris; 3, medial rectus; 4, lateral rectus; 5, superior oblique; 6, reflected tendon of superior oblique; 7, annulus of Zinn; 8, optic nerve; 9, ophthalmic artery; 10, inferior rectus; 11, inferior oblique.

general action of rotating the eye in the four cardinal directions—up, down, out and in (Fig. 356). They arise in a fibrous ring around the optic foramen to the nasal side of the axis of the eye and run to be inserted in the sclera by flat tendinous insertions about 10 mm. broad. The medial rectus is inserted into the sclera about 5.5 mm. to the nasal side of the corneo-scleral margin, the inferior rectus 6.6 mm. below, the lateral rectus 7 mm. to the temporal side, and the superior rectus 7.75 mm. above (Fig. 358). The *oblique muscles*, the primary function of which is rotation of the globe, are differently arranged (Fig. 357). The superior oblique arises from the common origin at the apex of the orbit, runs forwards to the trochlea, a cartilaginous ring at the upper and inner angle of the orbit and,

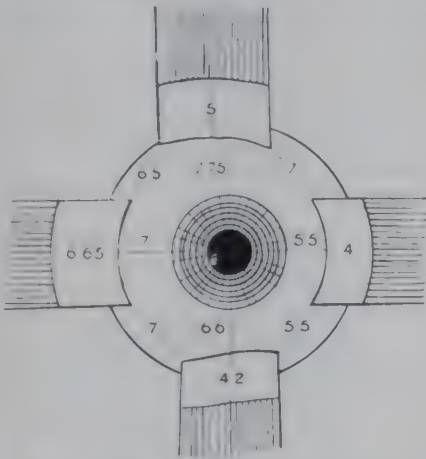


FIG. 358. Lines of insertion of the rectus muscles of right eye seen from the front.



FIG. 359. Lines of insertion of the superior and inferior oblique muscles and of the superior, medial and lateral recti of right eye, seen from above.

having threaded through this, becomes tendinous; the tendon changes its direction completely and runs over the globe under the superior rectus to attach itself above and lateral to the posterior pole (Fig. 359). The action of the muscle is thus determined by the oblique direction of its tendon after it has left the trochlea. The inferior oblique maintains a similar direction throughout its course and is the only muscle not rising from the apex of the orbit. It arises anteriorly from the lower and inner orbital walls near the lacrimal fossa and, running below the inferior rectus, finds an insertion in the sclera below and lateral to the posterior pole of the globe.

In order to control their movements all these muscles are provided with fascial *check ligaments* intimately connected with Tenon's capsule and the periosteum.

The Action of the Extra-ocular Muscles is to rotate the eye around a *centre of rotation*, which lies in the horizontal plane some 12 or 13 mm. behind the cornea, and in every movement of the globe every muscle is involved in some degree, either by

contraction or inhibition. Three types of rotation or "degrees of freedom" are possible around the centre of rotation :—

- (1) rotation around the vertical axis whereby the globe is turned from side to side,
- (2) rotation around the horizontal axis whereby the globe is turned upwards and downwards,
- (3) rotation around the antero-posterior axis—an involuntary movement of *torsion* : intorsion when the upper pole of the cornea rotates nasally, extorsion when temporally.

When the medial or lateral rectus acts it rotates the eye horizontally inwards or outwards. Owing to the obliquity of their course, however, contraction of the superior and inferior recti must involve torsion (Figs. 360-1). Thus, when the superior rectus acts upon

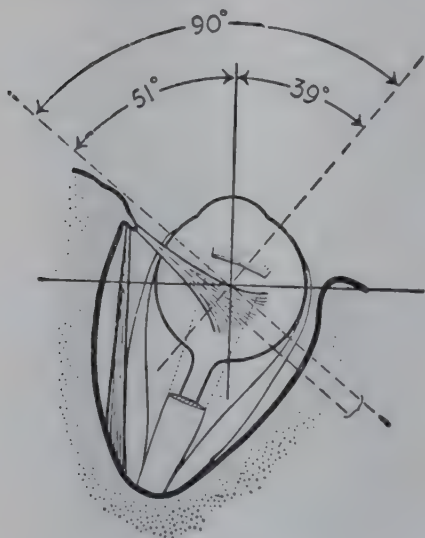


FIG. 360.

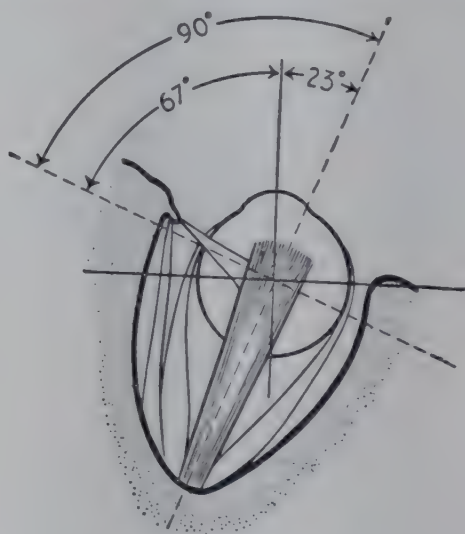


FIG. 361.

FIGS. 360-1. The effectivity of the vertical rotators in elevation. When the visual axis lies in the muscle plane, rotation upwards will be maximal ; in this position there is one direction of movement only—upwards and downwards ; the further the visual axis is removed from this position, the more effective the muscle becomes in torsion. Fig. 360. The superior (or inferior) oblique is 100 per cent. effective in depression (or elevation) when the eye is adducted 51° . It is ineffective when the eye is abducted 39° . Fig. 361. The superior (or inferior) rectus is 100 per cent. effective in elevation (or depression) when the eye is abducted 23° . It is ineffective when the eye is adducted 67° . In the primary position three-quarters of its efficiency is devoted to vertical rotation and one-quarter to torsion.

the globe in the primary position, it not only pulls the eye upwards, but also inwards and intorts it. Similarly when the inferior rectus acts the eye is pulled down and in and extorted. Since the obliques are inserted behind the centre of rotation, their direction of action is from behind forwards and inwards. When the superior oblique

contracts, therefore, if the globe is in the primary position the main effect is intorsion, but it also rotates the eye downwards and outwards; the inferior oblique causes primarily extorsion but rotates the globe upwards and outwards. The mechanism is so arranged that when the superior rectus and inferior oblique act simultaneously the eye moves directly upwards, the upward movement caused by each muscle being summated, while the inward movement and torsion of the superior rectus are exactly compensated by the outward movement and contrary torsion of the inferior oblique. Similarly when the inferior rectus and superior oblique act simultaneously the eye moves directly downwards.

Every movement of the eyeball is thus a synkinesis.

Not only is there uniocular synkinesis: in normal circumstances there is always also binocular synkinesis. Abduction of one eye is accompanied by adduction of the other—conjugate deviation; elevation or depression of one eye is always accompanied by elevation or depression respectively of the other. The only exception to this rule is the bilateral adduction of the eyes in convergence. Elevation of both eyes is accompanied by slight abduction (divergence), depression by slight adduction (convergence).

In these movements the muscles which contract together are called *synergists*; those which suffer inhibition, *antagonists*. The correlation may be summarized thus:

In rotation to the right—synergists: R. lat. rectus, L. med. rectus.

Antagonists: R. med. rectus, L. lat. rectus.

In rotation to the left—synergists: L. lat. rectus, R. med. rectus.

Antagonists: L. med. rectus, R. lat. rectus.

In rotation upwards—synergists: R. and L. superior recti primarily, R. and L. inf. obliques secondarily. Antagonists: R. and L. inf. recti, R. and L. sup. obliques.

In rotation downwards—synergists: R. and L. inf. recti primarily, R. and L. sup. obliques secondarily. Antagonists: R. and L. superior recti primarily, R. and L. inf. obliques secondarily.

The **nervous control of the ocular movements** is complicated. The muscles are supplied by nerves arising from nuclei in the mid-brain. Their action is co-ordinated by intermediate "centres" situated in this region by which reflex activities are governed. Finally these intermediate centres are linked with the vestibular apparatus whereby they become associated with the equilibration reflexes, and also with the cerebral cortex so that voluntary movements and participation in the higher reflexes involving perception become possible (Fig. 365).

The oculomotor, or third cranial nerve, supplies all the extrinsic muscles except the lateral rectus and superior oblique; it also supplies the sphincter pupillæ and ciliary muscle. The superior oblique is supplied by the fourth (trochlear) nerve, and the lateral rectus by the sixth (abducens) nerve.

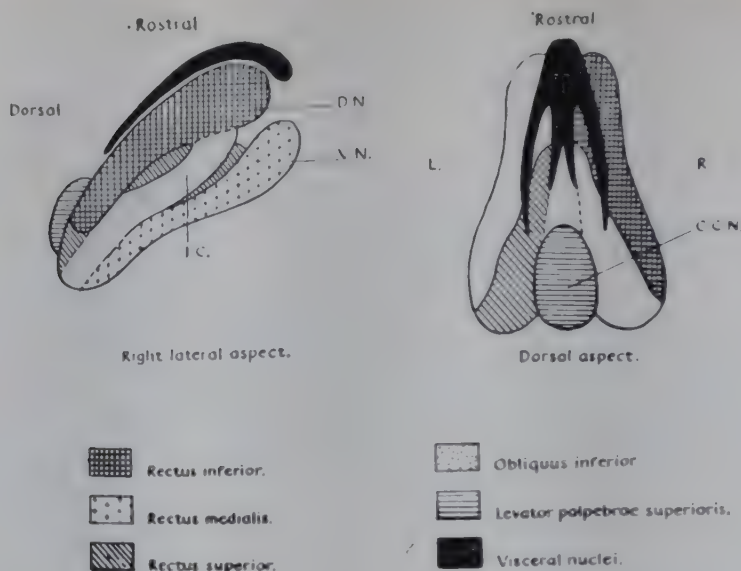


FIG. 362. The constitution of the third nerve nucleus, divisible into five cell-masses. DN, dorsal nucleus; VN, ventral nucleus; IC, intermediate cells; CCN, central caudal nucleus. The autonomic nuclei of Edinger-Westphal and Perlia are in black (after Warwick).

The third and fourth nuclei form a large continuous mass of nerve cells situated near the middle line in the floor of the aqueduct of Sylvius beneath the superior colliculus (Fig. 363). The cells nearest the middle line towards the anterior part of the third nucleus are smaller than the others: they form the Edinger-Westphal (and Perlia's) nucleus which probably supplies fibres to the ciliary muscle (accommodation) and sphincter pupillæ (constriction of the pupil). The main mass of the large-celled nucleus is composite, divided into cell-masses subserving the individual extrinsic ocular muscles as is seen in Fig. 362. The levator palpebrae is represented most caudally, while abduction is relegated to the sixth nucleus, situated much farther caudally in the brain-stem. There is little decussation of the fibres from the third nuclei of the two sides in their anterior parts, but a considerable amount in the posterior part.

The fourth nerve is unique among motor nerves in having a dorsal decussation. Nearly, if not quite all the fibres decussate in the superior medullary velum and are distributed to the superior oblique muscle of the opposite side.

The sixth nucleus is in the immediate vicinity of the facial (seventh) nucleus (Fig. 363), the fibres from which make a large bend around it. Hence vascular and other lesions of the sixth nucleus are very liable to be accompanied by facial paralysis on the same side. All the fibres of the sixth nerve are distributed to the ipsilateral lateral rectus.

The peculiarities of distribution of the fibres from the third, fourth and sixth nuclei to muscles partly on one side and partly on the opposite side of the body show that the nervous mechanism of co-ordination of these muscles is extremely complex.

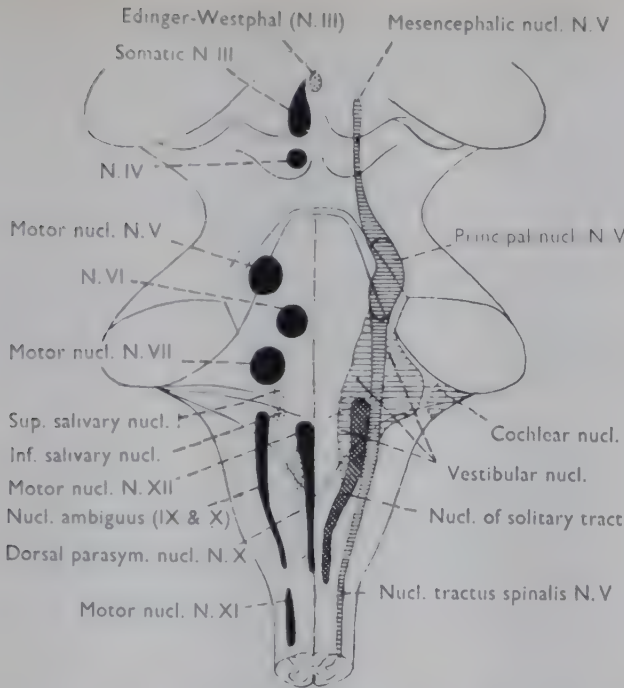


FIG. 363. The cranial nerve nuclei in the brain-stem.

In general the nuclei which are related to a particular type of fibre are arranged as a longitudinal column. The *motor nuclei* (drawn black) are arranged in 3 columns in a medial area. Those supplying muscles of myotomic origin—III, IV, VI and XII—are situated close to the midline beneath the floor of the 4th ventricle and the aqueduct. Nuclei innervating the branchial musculature—the motor nuclei of V and VII, and the nucleus ambiguus of IX and X—are arranged in a more lateral column and migrate ventrally during foetal development. The *visceral efferent nuclei* (seen dotted)—the Edinger-Westphal nucleus of III, the lacrimal, superior and inferior salivary nuclei belonging to VII and IX, and the dorsal motor nucleus of X—are more lateral still, and remain immediately beneath the floor of the ventricular system.

The *somatic afferent nuclei* (horizontal shading) occupy a lateral area; most medially are the 3 sub-divisions of the sensory nucleus of V, and more laterally the special somatic afferent nuclei of VIII—cochlear and vestibular. The *visceral afferent nucleus* of the solitary tract is seen cross-hatched. (After A. Brodal.)

The *intermediary mechanism* co-ordinating the activities of these nuclei is also complex. The nuclei are inter-connected to a considerable extent by fibres participating in the *posterior longitudinal bundle* (Fig. 366), a large and important tract of nerve fibres derived in part from the anterior columns of the spinal cord, lying in close relation to the third, fourth and sixth nuclei. These fibres probably have important functions in the co-ordination of movements and equilibration, which are so intimately related with vision. The nuclei are also inter-related through this bundle so that co-ordination of the two eyes is maintained. One of the most impor-

tant of such connections is the fibres which link up the sixth nucleus of one side with the third nucleus of the other in some such manner as depicted in Fig. 364.

In this region there are also "centres" which control binocular movements (Fig. 365).

There is probably an area for lateral movements in the neighbourhood of the sixth nucleus; the evidence in favour of the existence of an area controlling vertical movements is conflicting; the centre for convergence is associated with the third nerve nucleus and almost certainly lies in the Edinger-Westphal complex; and an area co-ordinating divergence possibly exists.

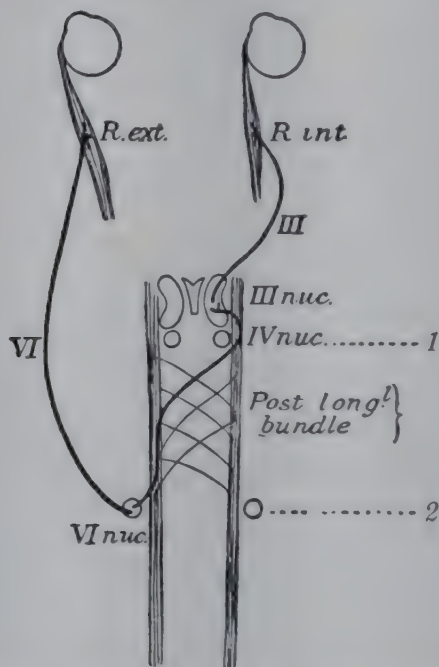


FIG. 364. Diagram of the course of the fibres from the sixth nucleus which are concerned in conjugate deviation of the eyes. 1 and 2, lines of section through the mesencephalon and the pons.

This elaborate mechanism in the mid-brain is controlled from three sources, one voluntary and three reflex.

(1) *Voluntary ocular movements* are initiated in the pyramidal cells of the motor area of the frontal cortex in the second and third frontal convolutions of both sides (Fig. 365). The fibres enter the knee of the internal capsule as part of the pyramidal tract close to the fibres governing facial movements and break off in the mid-brain, first the fibres for vertical movements (and movements of the upper lid) and then those for lateral movements. These fibres control conjugate movements, vertical and horizontal, of both eyes ; movements of individual muscles are not represented in the cortex. Stimulation of the cortex or the tracts therefore produces conjugate movements of the eyes to the opposite side ; destruction, paralysis

of voluntary conjugate movements away from the side injured, in either case without involving disalignment of the eyes or diplopia.

(2) The involuntary reflexes which depend on vision (fixation, fusional movements, convergence, etc.)—the *psycho-optical reflexes*—are centred in the visual cortex of the occipital lobe (Fig. 365). The afferent path for these reflexes is the visual pathway; the

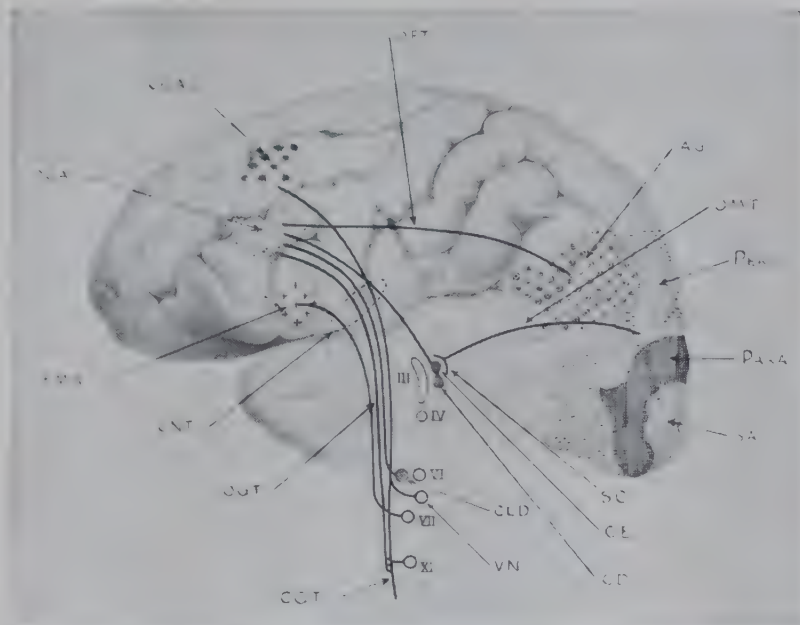


FIG. 365. The cerebral oculomotor connections. The elaborate connections of the ocular motor nuclei with the intermediary centres and with the visual and labyrinthine centres; proprioceptive afferent impulses are omitted. AG, angular gyrus; CD, centre for depression; CE, centre for elevation; CGA, cephalo-gyric area; CGT, cephalo-gyric tract; CLD, centre for lateral deviation; CNT, cortico-nuclear tract; FMA, facial muscle area; OFT, occipito-frontal association tract; OGA, oculo-gyric area; OGT, oculo-gyric tract; OMT, occipito-mesencephalic tract; PARA, parastriate area; PERI, peristriate area; SA, striate area; SC, superior colliculus; VN, vestibular nucleus.

efferent runs down the optic radiations to the posterior longitudinal bundle (Fig. 366) and thence to the ocular motor nuclei.

(3) An elaborate system of *stato-kinetic reflexes* of great phylogenetic antiquity co-ordinates the position of the eyes when the head is moved in space; their afferent path runs from the semicircular canals of the inner ear to the mid-brain centres. They induce conjugate movements of both eyes, a slow tonic movement in the direction of equilibration and a quick return (nystagmus).

(4) A similar system of *static reflexes* co-ordinates movements of the eyes in respect of movements of the head upon the body. They are mediated mainly by proprioceptive impulses from the neck muscles which are linked with the ocular motor centres through the posterior longitudinal bundle.

Of these, two important mechanisms deserve special mention at this stage—fixation and convergence.

FIXATION AND BINOCULAR VISION

We have already seen that the image of an external object on the retina is determined by a line passing from the object through the nodal point of the eye. Conversely an object is *projected* in space along the line passing through the retinal image and the nodal point. When a distant object is looked at, the visual axes are practically parallel; the object forms an image upon each fovea centralis. Any other object to one side forms its retinal images upon the temporal side of one retina and upon the nasal side of the other; these retinal areas are co-ordinated visually in the occipital cortex so that such an object is seen with both eyes as a single object. These are known as *corresponding points*, the most important pair of which, of course, is the foveæ. Points on the two retinae which are not corresponding points in this sense of the term are called *disparate points*, and if an object forms its retinal images thereon it will be seen double (binocular diplopia). If the disparity is slight there is a tendency to move the eyes so that the images may be fused by means of the fusion reflexes from the occipital cortex.

Since the most accurate vision is attained by the foveæ it is necessary that the eyes be rapidly orientated so that the image of an object of interest falls upon them or that of a moving object be retained on them. This ascendancy of the foveæ is maintained by the *fixation reflex* (Fig. 366) and whenever the image leaves the foveæ, the eyes are at once reorientated so that it falls upon them. The activity of this reflex is demonstrated by the rapid to-and-fro movements of the eyes of a person looking out of the window of a moving train, and can be demonstrated clinically by regarding a revolving drum on which black and white stripes are painted (*optokinetic nystagmus*). The latter phenomenon can be used as a test to demonstrate the integrity of the reflex paths.

The same reflex produces *involuntary fusional movements* of the eyes to maintain single binocular vision. They may be demonstrated clinically by placing a small prism in front of one eye while the patient regards a distant light. The eye will at once turn out of the primary position to allow the deflected rays to fall again upon the fovea (Fig. 366). The strongest deviation which can thus be overcome is a measure of the *reflex fusional capacity* (Figs. 369-70).

Binocular Vision. In view of the distance between the two eyes it is obvious that the retinal images of both eyes cannot be identical since each eye regards a slightly different aspect of any object observed. If the object is a solid body the right eye sees a little more of the right side of the object, and *vice versa*. The two images are fused psychologically, and this fusion of the slightly

diverse images, combined with other facts derived from experience, enables the person to appreciate the solidity of objects.

Moreover, it is obvious from Fig. 367 that if an object is regarded, the images of other objects nearer or further away cannot fall upon

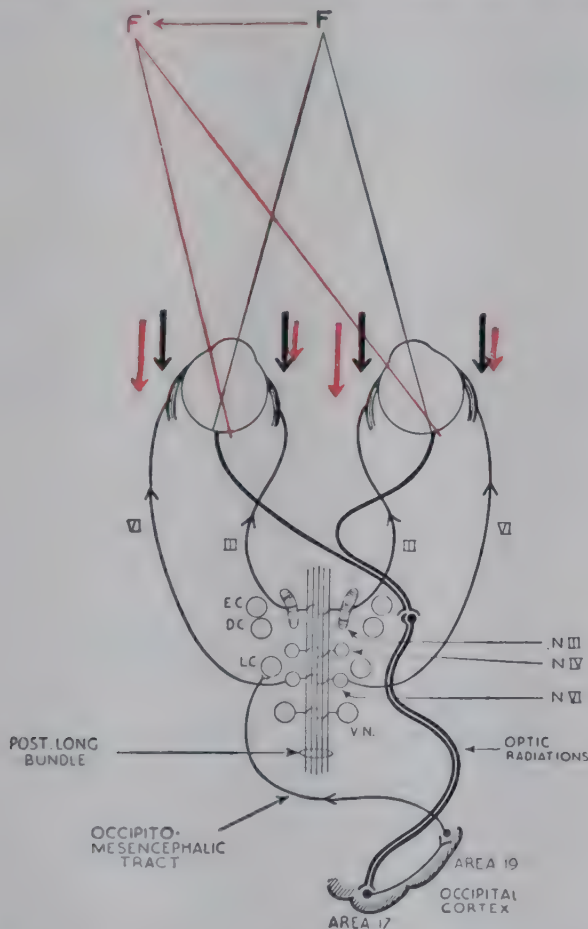


FIG. 366. The organization of the psycho-optical reflexes (fixation, refixation, fusional, etc.). So long as the fixation point (F) is imaged on each macula, the fixation reflex maintains the posture of the eyes steady by an equality of muscular tone (black arrows). If, however, F is moved to F' , the retina on the right of the macula is stimulated and sets up a refixation reflex. The afferent path is: (a) retina—optic nerves—chiasma—right optic tract; (b) lateral geniculate body—right optic radiations—striate area of occipital cortex; (c) peristriate occipital cortex. The efferent path is conjectural, but traverses the posterior part of the optic radiations and reaches the opposite cerebral peduncle where connection is made with the vestibular nucleus (VN), the mid-brain ocular motor centres governing conjugate elevation, depression and lateral movements (EC, DC, LC), whence impulses are relayed through the posterior longitudinal bundle to the ocular motor nuclei (III, IV, VI). In the present case, acting essentially through the left VIth and the branch of the right IIIrd to the medial rectus, the muscular tone is altered (red arrows) to orientate the eyes so that F' again falls on each macula.

corresponding points. If their projection through the nodal point is continued to the retina, it is seen that the images of objects nearer



FIG. 367. To illustrate physiological binocular double vision. For the sake of clarity the distances are out of proportion. The images of the fixation point (F) fall on each fovea (f); those of an object near the eye (T) will fall on t , giving rise to crossed diplopia.

than the object of fixation fall on the temporal side, those farther away to the nasal side of the fovea. This can be easily demonstrated

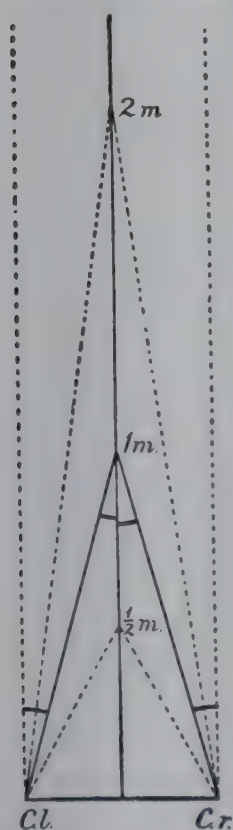


FIG. 368. Diagram of the metre angle. Cr., Cl., centres of rotation of the right and left eyes.

by holding a pencil in front of the eyes: if the pencil is fixated, more distant objects appear doubled; if a distant object is fixated, the pencil appears doubled. It will be found in this way that near objects suffer a *crossed (heteronymous) diplopia*; distant objects an *uncrossed (homonymous) diplopia*. This diplopia is physiological and is perceptually suppressed in actual vision, but it produces a psychological impression which is translated into appreciation of distance. It follows that accuracy of stereoscopic vision depends upon good sight with both eyes simultaneously.

CONVERGENCE AND ACCOMMODATION

When a distant object is observed by an emmetropic person the visual axes are parallel and no effort of accommodation is made. If, however, a near object is regarded, the eyes converge upon it and an effort of accommodation corresponding to the distance of the object is made. These movements are reflex and are controlled, as we have seen, by a centre in the occipital cortex (Fig. 365), the afferent path being the visual pathways, the efferent path running through the pontine centre which controls lateral ocular movements to the Edinger-Westphal nucleus. The associated pupillary contraction is

a purely low-level reflex arc, the afferent path running from the medial recti to the Edinger-Westphal nucleus and the efferent by the parasympathetic fibres in the third nerve (Fig. 36).

Convergence is usually measured by employing the *metre angle* as a unit. Suppose an object to be situated in the median line between the two eyes at a distance of one metre from them. Then the angle which the line joining the object with the centre of rotation of either eye makes with the median line is called one metre angle (Fig. 368). With an interpupillary distance of 60 mm. this angle is about 2° . If the object is two metres away the angle is approximately half as great, or $\frac{1}{2}$ m.a. If the object is 50 cm. away the angle

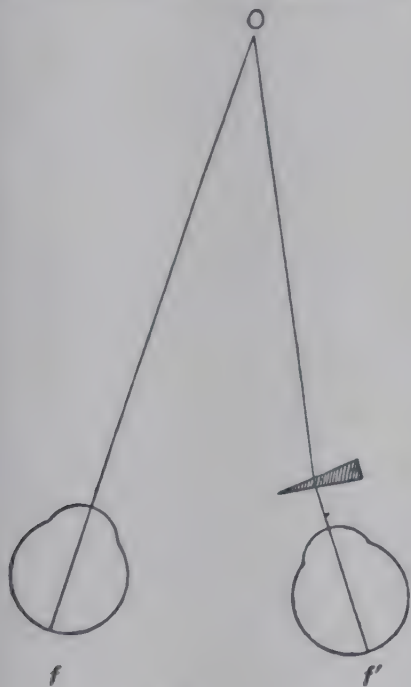


FIG. 369.

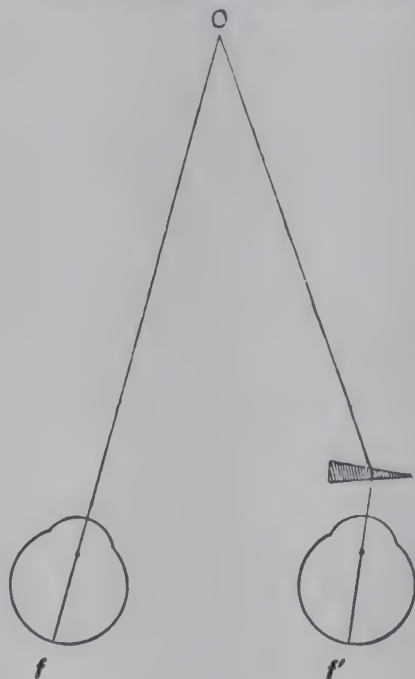


FIG. 370.

FIGS. 369-70. Diagrams of the action of adducting and abducting prisms. O, object of fixation; f , f' , left and right foveæ centrales.

will be 2 m.a. Now, the amount of accommodation which an emmetropic eye exercises in order to see clearly an object 1 m. away is 1 D, 2 m. away 0.5 D, 50 cm. away 2 D, etc. Hence with an emmetropic person the amount of convergence, reckoned in metre angles, is the same as the amount of accommodation reckoned in dioptries. Just as the difference in the amount of accommodation between the far point and the near point is called the amplitude of accommodation, so the difference in convergence between the far point and the near point is called the *amplitude of convergence*.

Clinically, convergence can be tested roughly by making the patient fix a finger or pencil which is gradually brought nearer to the eyes in the middle line. The eyes should be able to maintain convergence when the object is 8 cm. ($3\frac{1}{2}$ inches) from the eyes. If outward deviation of one eye occurs before this point is reached the power of convergence is deficient.

More accurate measurements can be made by instruments held in front of the eyes wherein a vertical pointer slides along a scale. The

amount of convergence can also be measured by prisms, as an extension of the method described to measure fusional capacity (Figs. 369-70). In this case the prism is directed base outwards; the strongest prism which can thus be used without inducing diplopia when a *distant* object is regarded is a measure of the amplitude of convergence.

Although related, it is obvious that the association between convergence and accommodation must be elastic—otherwise a hypermetrope whose accommodation is always used in excess would always have diplopia, or a presbyope who could not accommodate would be unable to converge. If a person fixates (and accommodates for) a *near* object, the amount of *positive convergence* is measured by the strongest prism, base out, which can be borne without causing diplopia (Fig. 369); the amount of *negative convergence* (or relative divergence) by the strongest prism, base in (Fig. 370). The amplitude of convergence therefore consists of a negative portion and a positive portion which vary with each distance of the object fixated.

The convergence synkinesis is so co-ordinated that the energy exerted is accurately divided between the two medial recti. Hence it is found that the effect is the same in the above experiments whether the prism is placed before only one eye, or a prism of half the strength is placed before each eye.

CHAPTER 29

PARALYTIC AND KINETIC STRABISMUS : SYNKINESES : NYSTAGMUS

Strabismus (στραβός, crooked) or **squint** is a generic term applied to all those conditions in which the visual axes assume a position relative to each other different from that conforming to physiological conditions. We have seen that the motility of the eyes is controlled by voluntary and reflex mechanisms centred in the cerebral cortex. Lesions at this level or in the supranuclear pathways produce *conjugate deviations* or *pareses* which affect both eyes equally ; although their movements or positions are abnormal, they maintain their relative co-ordination and diplopia is not produced. Simple ptosis, however, may result from such a lesion. The clinical incidence of these deviations will be discussed in Chapter 34 as well as the mixed palsies resulting from lesions in the mid-brain. If, however, the lesion is situated at the level of the lower neurons, affecting the nuclei, the nerves or the muscles, the relative co-ordination of the eyes is disturbed and diplopia and other symptoms appear. The usual result of such a lesion is paralysis (*paralytic squint*) ; sometimes it is due to irregular or spasmodic activity of individual muscles or groups of muscles (*kinetic squint*). We shall see presently that a further type of squint exists when the visual axes, although abnormally directed, retain their relative position in all movements of the eyes ; this is therefore termed *concomitant squint*.

PARALYTIC STRABISMUS

Signs and Symptoms. The signs and symptoms of paralytic squint comprise limitation of ocular movements, diplopia, false orientation, abnormal position of the head and vertigo.

(1) *Limitation of Movement.* In paralysis of an ocular muscle the ability to turn the eye in the direction of the normal action of the muscle is diminished or lost. In slight paresis the defect in mobility may be so small as to escape observation without special tests. In all positions in which the affected muscle is not brought actively into play the visual axes assume their normal relationship.

Limitation of movement is tested roughly by fixing the patient's head and telling him to follow the movements of the surgeon's finger. The finger should be held vertically in testing horizontal movements, horizontally in testing vertical movements. An accurate record of the movements of each eye can be obtained by taking the *field of fixation* with the perimeter as for recording the field of vision. With the head immobile and the other eye screened the patient looks as far as possible

along the arc of the perimeter, test types being moved in from the periphery until he is just able to read them. The normal field of fixation is about 50° downwards and 45° in all other directions.

The relative movements of the two eyes when each is used for fixation are of importance. This is most readily explored by the *screen test*. When the eyes are turned in the direction of the normal action of the paralysed muscle movements of the affected eye are impeded. It therefore deviates relatively to the other eye; this

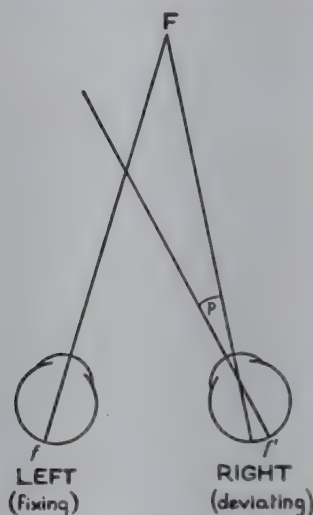


FIG. 371.

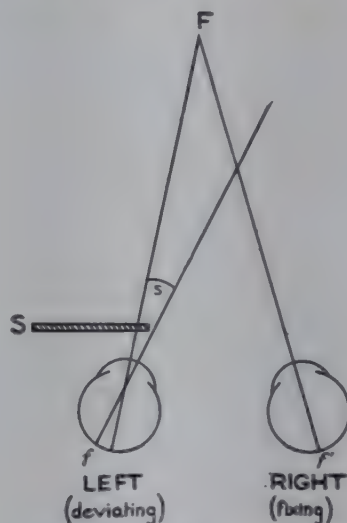


FIG. 372.

FIGS. 371-72. Primary and secondary deviations. In concomitant squint (affecting the right eye) the primary deviation (p , Fig. 371) is equal to the secondary deviation (s , Fig. 372) obtained when the non-squinting eye is occluded by a screen, S. F, fixation point. In paralytic squint the secondary deviation is greater than the primary.

position is called the *primary deviation* (Fig. 371). The angle of deviation is the angle which the line joining the object of fixation and the nodal point makes with the visual line.

If, on the other hand, the paralysed eye is used for fixation it will have difficulty in moving in this direction; since the nervous energy required for movement is equally distributed between the two, the normal eye will share in this abnormal effort. If, therefore, the sound eye is covered by a screen, and an attempt is made to fix an object so situated that the paralysed muscle is brought into play, it will be found that the normal eye behind the screen deviates more than the primary deviation of the paralysed eye. For example, if the right lateral rectus is paralysed and the left eye is covered, then on attempting to fix an object situated to the right with the right eye, the left eye will deviate very much to the right, so much in fact that its line of vision is well to the right of the object fixed. Hence, if the screen is removed suddenly the left eye will spring back

to the left so as to take up fixation. This deviation of the sound eye is called the *secondary deviation* (Fig. 372). This feature is of great importance because when well marked it distinguishes paralytic squint from the concomitant type in which the secondary deviation is equal to the primary.

(2) *Diplopia*. The chief complaint of patients with paralysis of an extrinsic muscle is usually that they see double. Diplopia occurs only over that part of the field of fixation towards which the affected muscle or muscles move the eye. If both eyes are functional and one deviates, *binocular diplopia* results.

The image seen by the squinting eye (*false or apparent image*) is usually less distinct than that seen by the fixing eye (*true image*), because only in the latter case does the image fall upon the fovea centralis. The angular displacement of the false image is equal to the angle of deviation of the eye.

Suppose the left eye fixes accurately while the right deviates inwards, a bright, sharply defined foveal image is seen with the left eye. The image formed by the object on the right retina, falling as it does upon the line joining the nodal point with the object, lies to the nasal side of the retina. The patient, being unconscious of the malposition of his eye, orientates the object subjectively as if the eye were straight. He knows from experience that objects which form their images upon the nasal side of the retina are situated to the temporal side. He therefore projects the object with this eye to the right of its actual position. This is called *homonymous diplopia*, because the object as seen by the right eye is to the right of the object as seen by the left eye (Fig. 373). If the right eye deviates outwards, *heteronymous* or *crossed diplopia* results, because the object as seen by the left eye lies apparently to the right of the object as seen by the right eye (Fig. 374).

(3) *False Orientation*. It will be seen from what has already been said that false orientation is a necessary accompaniment of binocular diplopia. Suppose that a patient whose right lateral rectus is paralysed shuts his left eye and attempts to fix an object situated towards the right. Let him now quickly point at the object with his extended index finger; the finger will pass considerably to the right of the object. This is called *false projection*. It depends upon the same principle as the increase of the secondary deviation, for the object is projected according to the amount of nervous energy exerted; as this is greater than that exerted in normal circumstances, the object is projected too far in the direction of action of the paralysed muscle. It is essential that the finger should be directed at the object quickly, otherwise the error is noticed and compensation is made. For example, if in the same circumstances the patient is told to walk towards an object situated at some distance to the right, he first steps too far to the right, then recognizes his mistake and corrects it. In old paralysis the patient may learn by experience to compensate for the error.

(4) *Position of the Head.* The patient holds his head so that his face is turned in the direction of action of the paralysed muscle. For example, in paralysis of the right lateral rectus the patient keeps his head turned to the right. The object of this manœuvre is to lessen the diplopia and its attendant unpleasant consequences as much as possible. In complex paralysis the position of the head is still such as to relieve the diplopia to the maximum extent, the position being unconsciously adopted.

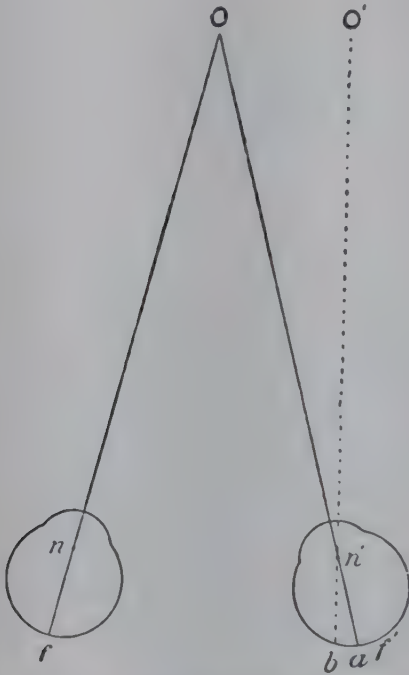


FIG. 373. Diagram of homonymous diplopia.

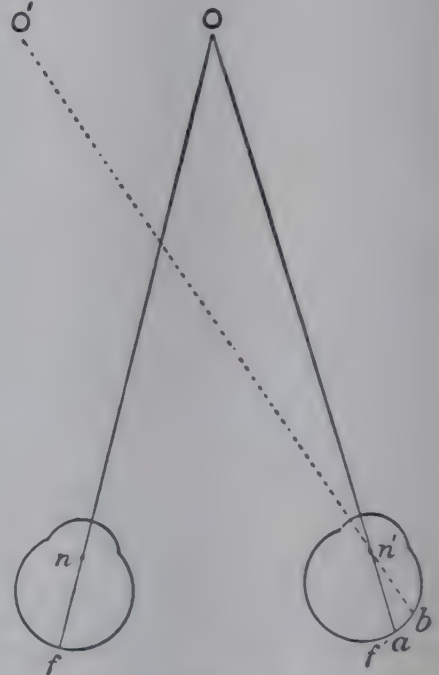


FIG. 374. Diagram of heteronymous (crossed) diplopia.

f, f' , left and right foveæ; n, n' , left and right nodal points. The image of O formed at a is projected as if a were the fovea, i.e., to O' from b .

"*Ocular torticollis*" is a term sometimes applied to tilting of the head to compensate for defective vertical movements of one eye. It is distinguished from true torticollis due to contraction of the sterno-mastoid muscle in that there is a simple tilting of the head without rotation of the chin towards the opposite shoulder; moreover, the sterno-mastoid is not unduly contracted. It occurs chiefly in cases of congenital origin—probably mal-insertion of the muscles—but has been met with after interference with the pulley of the superior oblique in frontal sinus operations. The vertical squint is made manifest by placing the head straight, when diplopia is also elicited.

(5) *Vertigo.* Vertigo, leading to nausea and even vomiting, is due partly to diplopia, partly to false projection. It occurs chiefly when the paralysed muscle is called upon to exert itself. When the

gaze is turned from the region of correct to that of false localization, objects appear to move with increasing velocity in the direction in which the eye is moving. The unpleasant symptoms are counteracted partially by altering the position of the head, or completely by shutting or covering the affected eye.

In congenital strabismus these symptoms are not obtrusive since the vision of one eye is suppressed or false retinal correspondences develop ; in these cases contraction of the antagonistic muscles does not occur. In acquired cases they are at first very distressing and incapacitating. In paralyses of long standing, however, relief is gradually obtained : false orientation and diplopia tend to disappear or become less troublesome, for the patient learns to ignore the impressions derived from the affected eye. Moreover, contraction of the antagonists of the paralysed muscle gradually sets in, which has the effect of increasing the deviation. Since the retinal image is thus thrown farther to the periphery of the retina where the sensitivity is less, its suppression is facilitated.

Investigation of a Case of Ocular Paralysis. The patient usually seeks advice on account of diplopia. In some cases the nature of the case is obvious immediately from the strabismus or from the manner in which the head is held. In most cases these features are too slight to decide the diagnosis and special tests must be made.

(1) The first procedure should be to cover one eye in order to determine whether the diplopia is unocular or binocular.

(2) If it is decided that the diplopia is binocular, the patient should fix the surgeon's finger, and the field of fixation of each eye should be carefully investigated. In cases of complete paralysis of one or more muscles it may be possible to make an accurate diagnosis from the observation of the defective movements combined with investigation of the exact positions of the images of the finger in different areas of the field of binocular fixation. In cases of paresis the differentiation of the images is too obscure to permit the solution of the problem by this means.

(3) In such cases the diplopia must be investigated by more delicate tests. In a dark room a red glass is placed before one eye and a green before the other in order to distinguish their images. A bar of light through a stenopæic slit in a hand-torch is then moved about in the field of binocular fixation at a distance of at least four feet from the patient, the patient's head being kept stationary. The positions of the images are accurately recorded upon a chart with nine squares marked upon it (Fig. 380). The examination may be carried out by the surgeon turning the patient's head in various directions while the light is kept stationary. The following data are derived from this examination :

- (a) the areas of single vision and diplopia ;
- (b) the distance between the two images in the areas of diplopia ;
- (c) whether the images are on the same level or not ;
- (d) whether one image is inclined or both are erect ;
- (e) whether the diplopia is homonymous or crossed.

These data, if concordant, are sufficient to diagnose the paralysis. The false image, which is frequently tilted and the fainter of the two, is determined by the direction in which the images are most separated from each other, in which case it is displaced farthest in the direction of the normal action of the paralysed muscle. By covering one eye it can be shown to which eye this image belongs.

The deviation of the false image is most easily determined when the eye is turned in the *cardinal positions*. For lateral palsies where only two muscles are involved, the test is easy ; greatest diplopia occurs in the horizontal line to the right in paralysis of the right lateral or left medial rectus, to the left for the left lateral or right medial rectus.

For vertical movements the action of four muscles must be analysed. They are most easily differentiated thus.

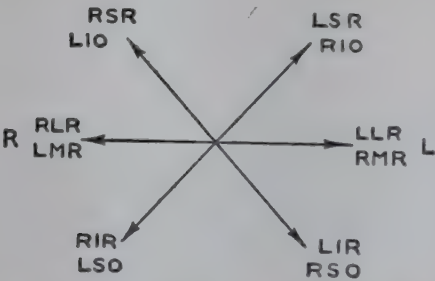


FIG. 375. The fields of action of the ocular muscles in binocular movements. The maximum effectivity as elevators is attained by the RSR and the LIO when acting as right-hand elevators.

<i>Rt. Sup. Area</i>	<i>Lt. Sup. Area</i>
R S R	L S R
L I O	R I O
R L R	L L R
L M R	R M R
<i>Rt. Inf. Area</i>	<i>Lt. Inf. Area</i>
R I R	L I R
L S O	R S O

FIG. 376. The mnemonic of Maddox for determining the paralysed muscle in the diplopia test (*cf.* Fig. 375).

In view of the obliquity of their course (p. 441) the recti are most effective as vertical rotators as the eyes are abducted from the primary position, the obliques when adducted. On looking up to the right the right superior rectus and the left inferior oblique are therefore primarily involved, the false image being higher than the true and tilted. The corresponding displacements in the four cardinal positions are seen in Fig. 375.

It must be remembered that these tests are purely subjective. In many cases the patients are uncooperative or their intelligence is obscured by intracranial disease ; or contracture of the antagonistic muscles may have set in. Consequently the answers are not infrequently discordant, and accurate diagnosis may be extremely difficult or impossible. As additional complications, the paresis may unmask a latent squint (p. 468), or the patient may fix with the paralysed eye, especially if this eye has the greater acuity of vision.

Considerable ingenuity has been used to devise mnemonics for determining the position of the false image. One of the most satisfactory is that of Maddox shown in the accompanying diagram (Fig. 376) ; if the field is divided into areas as shown, in vertical palsies the paresis is due to failure of the "same-named" rectus muscle (in the left superior area, the left superior rectus) or the most "crossed-named" oblique (right inferior oblique). In all cases the most peripheral image belongs to the palsied eye.

It may be pointed out that all the signs, with the exception of the deviation of the eye—defective movement, false projection, increase of diplopia

secondary deviation, and position of the head—are towards the side of the paralysed muscle.

To measure the degree of deviation, especially if torsional, and particularly to measure any progressive increase, the *Hess screen* (Fig. 377) is useful. It consists of a tangent screen marked in red lines on a black cloth with red spots at the intersection of the 15° and 30° lines with themselves and with the horizontal and vertical lines; over it three green threads are suspended in such a way that they can be moved over the screen in any direction by a

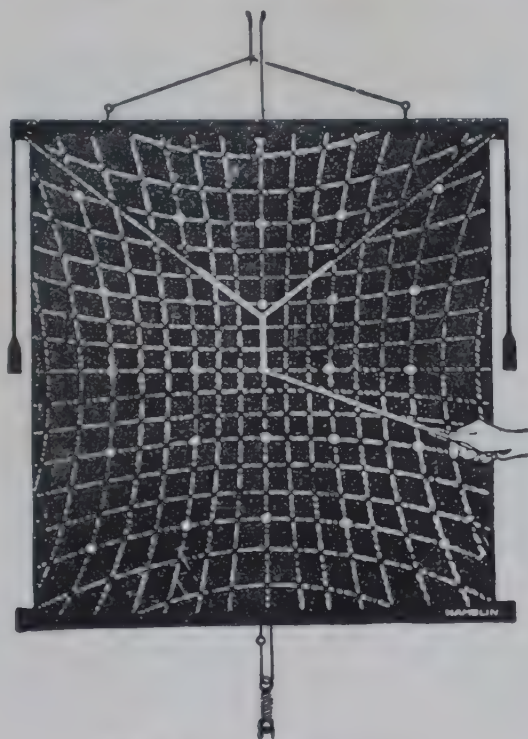


FIG. 377. Hess screen.

pointer. The patient, wearing red-and-green glasses, is asked to place the junction of the three threads over the red spots in turn. Through the red glass he can only see the red markers and through the green, the green threads, so that he indicates the point at which one eye is looking when the other fixes a spot. The position on which the indicator appears to coincide with the spot gives a permanent record of the primary and secondary deviation. The test also provides an accurate measure of comitance. In a concomitant squint the fields of each eye, although relatively displaced, are equal in area and undistorted; in parietic squint the area on the affected side is diminished away from the affected muscle and in spastic squint it is increased towards the affected muscle.

Varieties of Ocular Paralysis. If one muscle alone is affected it is generally the lateral rectus or the superior oblique, since each of these is supplied by an independent nerve. Affection of several muscles simultaneously is usually due to paralysis of the third nerve. All the extrinsic and intrinsic muscles of one or both eyes may be paralysed—*total ophthalmoplegia*. If only the extrinsic muscles are affected the condition is called *external ophthalm-*

moplegia ; if only the intrinsic (sphincter pupillæ and ciliary muscle) *internal ophthalmoplegia*.

Paralysis of the Lateral Rectus. There is limitation of movement outwards, and the face is turned towards the paralysed side. Homonymous diplopia occurs on looking to the paralysed side ; the images are on the same level and erect, becoming more separated on looking more towards the paralysed side. The false image is slightly tilted on looking up or down as well as towards the paralysed side (Figs. 378-80).



FIG. 378.



FIG. 379.

FIGS. 378-9. R. abducens palsy. Fig. 378, looking forwards ; Fig. 379, looking right.

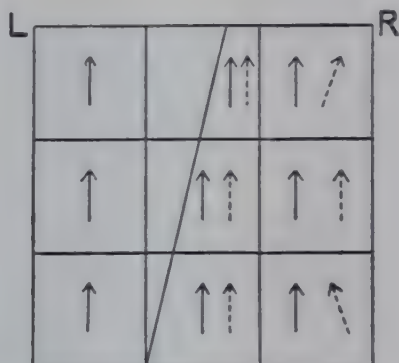


FIG. 380. Diplopia chart for the right lateral rectus. The oblique line through the chart shows the limit of the fields of single vision and of diplopia. The dotted arrows show the positions of the false image in different parts of the field of diplopia.

Paralysis of the Superior Oblique. There is limitation of movement downwards and towards the sound side ; the face is turned downwards and towards the *sound* side. Homonymous diplopia occurs on looking down (Fig. 382) ; the false image is lower and its upper end is tilted towards the true image. The distance between the images increases on looking down and towards the sound side and the inclination of the false image increases on looking down to the paralysed side. The patient has great difficulty in going downstairs, and vertigo is usually a particularly prominent symptom.

Paralysis of the Third Nerve. In complete paralysis of the third nerve there is ptosis, which prevents diplopia. On raising the lid with the finger the eye is seen to be deflected outwards and somewhat downwards, owing to the tone of the two unparalysed muscles. The pupil is semi-dilated and immobile, and accommodation is paralysed. There is a slight degree of proptosis, owing to loss of tone of the paralysed muscles. There is limitation of movements upwards and inwards, to a less degree downwards. With the lid raised there is diplopia, which is crossed, the false image

being higher, with its upper end tilted towards the paralysed side (Figs. 381, 383).

Paralysis of the third nerve is often incomplete, and individual muscles may occasionally be affected alone.

The *ætiology* of paretic strabismus is composite. The central nervous diseases which may entail a *lesion of the ocular motor nuclei* are discussed in Chapter 34. The most common cause is a small hæmorrhagic or thrombotic lesion in the mid-brain; syphilis used



FIG. 381. Right IIIrd paralysis. The position of strong attempted version to the sound side. The left eye is abducted, the right does not reach the midline. The right upper lid is raised to overcome the ptosis.

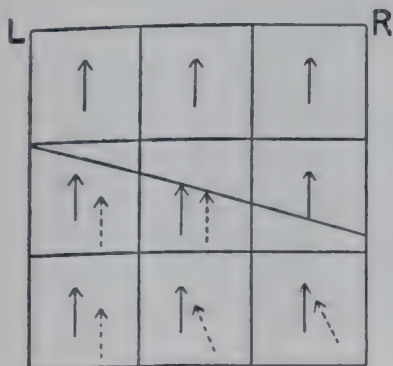


FIG. 382. Diplopia chart for paralysis of the right superior oblique.

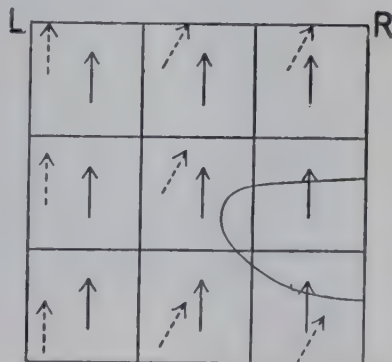


FIG. 383. Diplopia chart for the right third nerve. The area enclosed by the curved line is the area of single vision.

to be the most frequent *ætiological* factor. Other causes are infections of the central nervous system (encephalitis lethargica, poliomyelitis, etc.), toxins of endogenous (diphtheria) or exogenous (lead, botulism) origin, and thiamine deficiency.

Lesions of the nerve trunks are similarly common. They may be involved in infections of the meninges, cavernous sinus or orbit, by pressure from tumours, or trauma involving a fracture of the base of the skull. Among tumours, secondary invasion of the orbit or skull by malignant nasopharyngeal tumours (p. 526) should be remembered; of these, ocular palsies are a common early symptom. In pressure paralysis the nerve most frequently involved is the sixth; paralysis of the lateral recti is thus common in cases of intracranial tumours with high intracranial pressure, and generally has no localizing value. It may be due to traction on the nerves as

they bend over the apex of the petrous portion of the temporal bone (Figs. 384-5), or to pressure by the anterior inferior cerebellar and internal auditory arteries, which cross them at right angles and often lie ventral to them; the nerves are strangulated between the vessels and the œdematous and swollen pons. The paralysis, generally of the lateral rectus, following spinal anæsthesia may

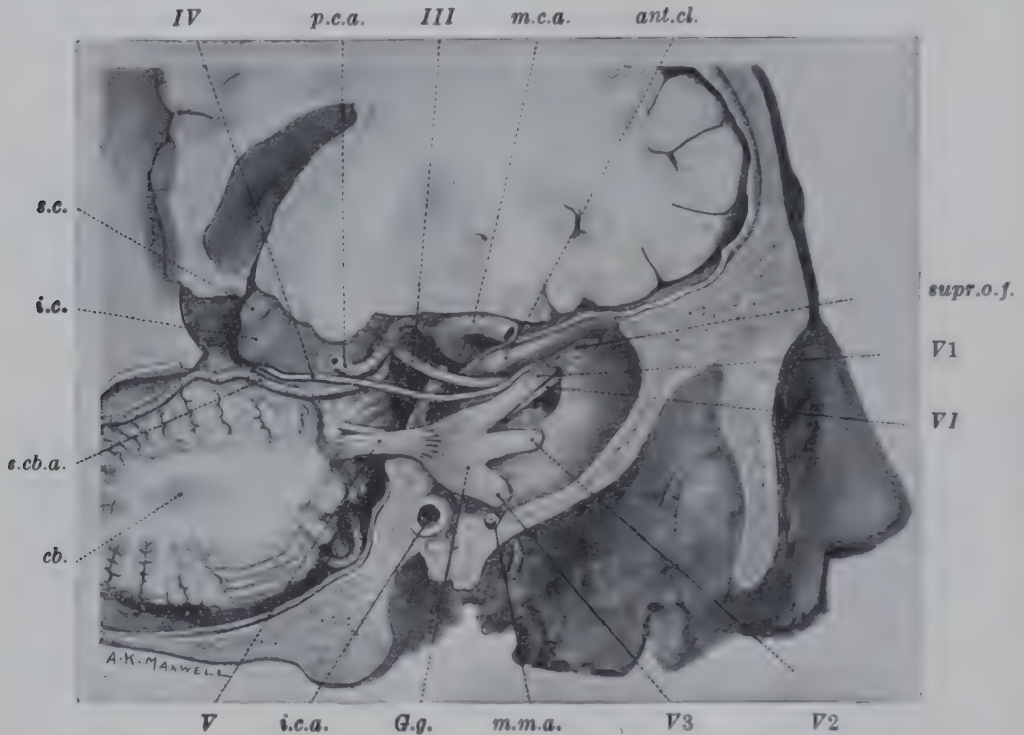


FIG. 384. Third, Fourth, Fifth and Sixth Nerves: *G.g.*, Gasserian ganglion; *i.c.a.*, internal carotid artery; *m.m.a.*, middle meningeal artery; *p.c.a.*, posterior cerebral artery; *m.c.a.*, middle cerebral artery; *ant.cl.*, ant. clinoid process; *supr.o.f.*, superior orbital fissure; *s.c.*, superior colliculus; *i.c.*, inferior colliculus; *s.cb.a.*, superior cerebellar artery; *cb.*, cerebellum. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

be due to the same cause; the onset is rapid, and recovery usually takes many weeks. Since the third nerve passes between the superior cerebellar and the posterior cerebral arteries, the same mechanism may account for ophthalmoplegic migraine (*q.v.*).

Paralysis of the lateral rectus, sometimes bilateral, is common in babies. It may be due either to pressure on the head at birth, a factor to which the sixth nerve is most exposed, or to maldevelopment of the nucleus.

Lesions of the muscles as well as of the branches of the nerves may occur with orbital disease or injury; the latter is relatively common. A palsy due to inefficiency of the superior oblique owing to dislocation of the trochlea is one of the more common lesions of this type.

Treatment. Aetiological treatment should be directed to the cause of the palsy, but effective measures are rarely possible.

Occasionally symptomatic treatment affords relief to the patient. The diplopia may sometimes be relieved by suitable prisms, but this treatment is rarely of much use owing to the variations in the amount of the deviation in different positions of the eyes. Occasion-

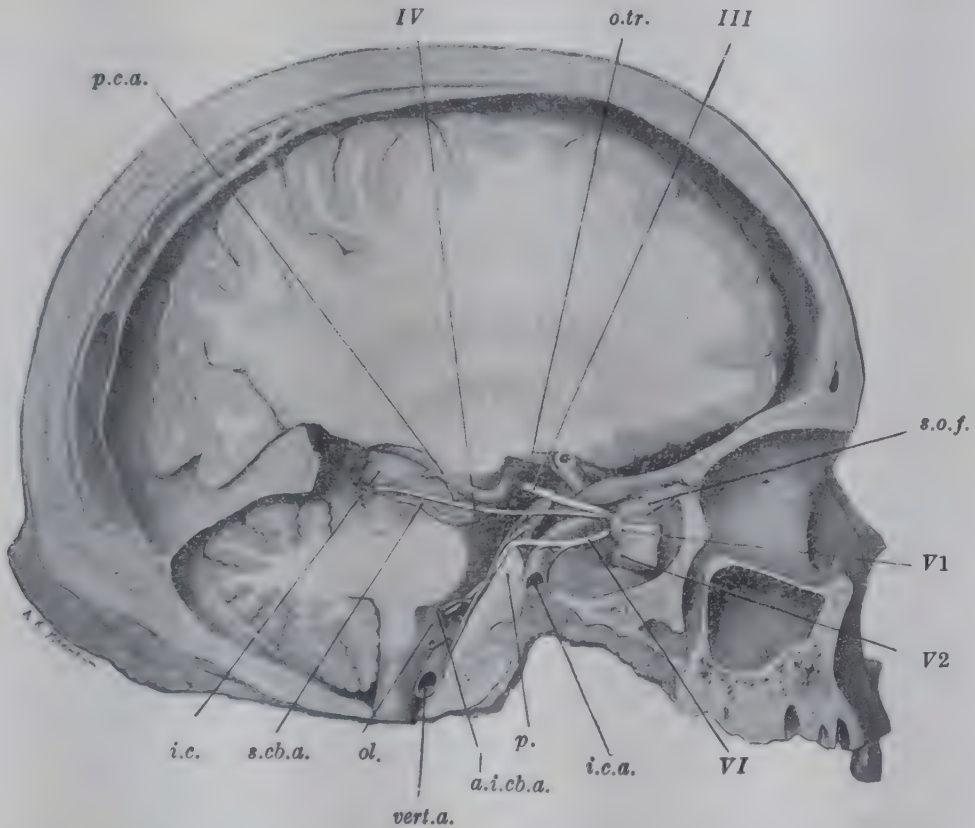


FIG. 385. Course of Sixth Nerve: *p.c.a.*, posterior cerebral artery; *o.tr.*, optic tract; *s.o.f.*, superior orbital fissure; *i.c.a.*, internal carotid artery; *p.*, petrous portion of temporal bone; *a.i.cb.a.*, anterior inferior cerebellar artery; *vert.a.*, vertebral artery; *ol.*, olive; *s.cb.a.*, superior cerebellar artery; *i.c.*, inferior colliculus. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

ally good is done by orthoptic exercises (p. 478). In old cases of partial paresis (never complete paralysis) an operation is indicated when the deviation has become stabilized—usually recession of the synergist muscle in the other eye to equalize the two, followed, if necessary, by recession of the antagonistic muscle in the same eye with advancement of the paralysed muscle, thus putting the affected muscle under better mechanical conditions. Those operations should always be done in stages to assess the effects of each; the techniques are described in Chapter 30.

If diplopia is very troublesome and cannot be relieved by the

means suggested, spectacles should be ordered with a ground glass in front of the affected eye or an opaque disc should be used.

Congenital strabismus is not uncommon. It is usually due to ineffectivity of a muscle owing to its mal-insertion, sometimes to a defect or absence of the nervous motor mechanism, sometimes to fibrosis of the muscle, and occasionally to absence of the muscle itself. Sometimes the defects are extensive and movements irregular or grossly deficient. More often one or a few muscles are involved, most commonly the lateral rectus, the superior rectus and oblique. If the defect is slight the squint eventually takes on the characteristics of a concomitant strabismus from which it is frequently difficult to differentiate in later life.

One of the common congenital defects is *Duane's retraction syndrome*. It is due to a fibrosis of the lateral rectus; in the primary position the eyes are straight or show some latent convergence, but a restriction or absence of abduction. On adduction there is a retraction of the globe and a narrowing of the palpebral fissure while on attempted adduction there is a slight exophthalmos. A second anomaly is the *superior oblique sheath syndrome* wherein there is a marked defect of elevation in the adducted position somewhat resembling the effects of a paresis of the inferior oblique muscle. In neither of these cases can the affected movement be passively accomplished, as by forceps when the patient is anæsthetized.

KINETIC STRABISMUS

Aberrant forms of strabismus occur as the result of irritative intracranial lesions, and are due, not to paralysis, but to irregular action or over-action of certain muscles, caused by unequal stimulation of the nerve centres or nerves. Such squints may occur in meningitis and lesions of the mid-brain or cerebellum, such as tumours (glioma, tubercle,



FIG. 386.



FIG. 387.

FIGS. 386-7. Overaction of the left inferior oblique. On deviation to the right (Fig. 386), the left eye shoots up and in; on deviation to the left (Fig. 387), the visual axes remain parallel.

gumma, etc.). The occurrence of the squint only during epileptiform fits or its irregularity of type may render the diagnosis from paralytic squint easy, especially when there are other prominent symptoms of cerebral irritation. In other cases, especially in the early stages of the disease, the diagnosis from paralytic or concomitant squint may be extremely difficult.

A second, more common, cause of such a squint is the spasmodic contracture which develops in the antagonist of a paretic muscle. The

muscle usually affected is the inferior oblique following a paresis of the superior rectus or superior oblique, frequently congenital in origin. The deviation is typical. On looking away from the affected side, as the eye is adducted and the inferior oblique comes into play, it is suddenly jerked up and in (Figs. 386-7). Treatment is by myectomy of the muscle (p. 482).

SYNKINESSES

The extrinsic muscles take part in many normal and pathological synkineses. When the eyes look up, the levatores palpebrarum raise the lids and in extreme upward movements the frontales also contract. In congenital ptosis (*q.v.*) upward movement of the eyes is often defective. On looking down the lid follows the globe; in exophthalmic goitre the lid follows tardily or not at all (*von Graefe's sign*); in total facial paralysis the lid follows the globe on looking down, although the eye cannot be closed voluntarily. On closing the lids, as in sleep, the eyes generally turn upwards and outwards (*Bell's phenomenon*). The same movement of the eyes occurs on attempted closure in total facial paralysis.

Other pathological synkineses are probably due to congenitally abnormal associations between two nerves or to aberrant regeneration of nerve fibres along the wrong nerve sheath after disease or injury. The "jaw-winking" *synkinesis* (of Marcus Gunn) is particularly striking. In these rare cases one levator palpebræ is thrown into spasm during eating, and sometimes on reading aloud. The upward lid movement is especially associated with lateral movements of the jaw, due to action of the pterygoid muscles which are innervated by the fifth nerve. In most cases, but not all, there is slight ptosis of the affected lid, and in cases with congenital ptosis the synkinesis occurs on sucking. Allied to the jaw-winking cases are others in which spasmodic lid movements occur on lateral deviation of the eyes.

The convergence pupillary synkinesis has already been mentioned: to it may be added the contraction of the pupil on forced closure of the lids. In rare cases spontaneous rhythmical variations in the size of the pupil are accompanied by ocular or lid movements. They are usually associated with congenital or early infantile paresis of the third nerve. Of these, a rhythmic *cyclic oculomotor spasm* is one of the most dramatic. In the mydriatic phase there is total ophthalmoplegia with ptosis, and at intervals of a minute or less a miotic phase develops in which the upper lid retracts, the eyes converge, the pupil contracts and the accommodation undergoes spasm.

NYSTAGMUS

Nystagmus (*νυστάγμις*, to nod) is the term applied to rapid oscillatory movements of the eyes, independent of the normal movements which are not affected. The oscillations are involuntary, although in rare cases normal persons can imitate them. They are usually lateral, but vertical, rotatory, and mixed rotatory and lateral or vertical nystagmus occur. The condition is almost always bilateral, although the movements may be much more marked in one

eye than the other. In such cases it may be necessary to examine the eye very carefully with the ophthalmoscope before the presence of nystagmus can be demonstrated in the magnified ophthalmoscopic image. Unilateral nystagmus does occur, but it is probable that many of the cases described are really bilateral. In *latent nystagmus* no movement is present when both eyes are open but nystagmus is elicited when either eye is covered.

Nystagmoid jerks—larger rhythmic jerking movements, most pronounced at the extreme limits of the normal movements of the eyes—should be distinguished from true nystagmus. They are not uncommon in normal people in certain conditions such as fatigue. The fundamental cause is probably quite different from that of true nystagmus, although both may occur together. *Optico-kinetic nystagmus* has already been noted (p. 448).

Nystagmus may be *congenital or early infantile*, or it may be *acquired*. These two groups of cases should be carefully distinguished on account of their different pathological foundations. Congenital and early infantile nystagmus dating from birth or within a few weeks of birth, occurs in congenitally malformed eyes, in albinism, and in eyes with congenital or early acquired opacities of the media (such as leucoma or anterior polar cataract due to ophthalmia neonatorum) or macular disease. The cause in these cases is inability to develop normal fixation. Fixation is normally developed during the first few weeks of life, the eyes being moved aimlessly and independently before it is acquired. Any cause operative at this period seriously diminishing the acuity of macular vision is liable to give rise to nystagmus; if the eye is blind, nystagmus does not develop but vague “searching movements” are seen. Nystagmus is present in most cases of total colour blindness (*q.v.*) in which vision is carried out by the rods alone, and there is therefore a central scotoma. In some congenital cases it is impossible to discover any cause. In a few such cases ancestors or relations have been albinos.

Nystagmus in infancy may be acquired after the period at which fixation is developed. This form occurs in the first year of life as *spasmus nutans*, in which it is associated with nodding movements of the head. The nodding of the head may be antero-posterior (affirmation), lateral (negation), or rotatory. It develops some weeks before the nystagmus, ceases during sleep, and disappears before the nystagmus. The nystagmus is very fine and rapid, and may be vertical, rotatory, or lateral and is generally more marked in one eye. The whole symptom-complex disappears in time—one of the few cases in which nystagmus spontaneously resolves. The nystagmus may disappear in one eye before the other; such cases may be mistaken for true unilateral nystagmus. In rare cases head nodding with nystagmus is congenital and hereditary, a condition which persists throughout life.

Nystagmus in adults occurs in diseases of the mid-brain, cerebellum and vestibular tracts, and of the semicircular canals. It is common

in multiple sclerosis wherein the movements are generally horizontal and are elicited in the early stages only in extreme lateral positions of the eyes. Cerebellar irritative lesions cause coarse nystagmus towards the side of the lesion and fine nystagmus to the opposite side. Nystagmus may also occur in adults as an "occupation neurosis," the commonest form being coal-miners' nystagmus (*vide infra*).

In congenital and early infantile nystagmus the patient is wholly unconscious of the movements, since objects do not appear to move. Vision is usually defective in spite of correction of errors of refraction which generally accompany the defect. In some cases of acquired nystagmus in adults, objects appear to move.

Labyrinthine Nystagmus occurs in disease of the internal ear in which the semicircular canals are involved, and can be produced in normal subjects by rotation in a specially designed chair, by syringing the ears or by passing a galvanic current through the head. The nystagmus is rhythmic, with a rapid and a slow component, is bilateral, and horizontal or rotatory, but varies according to the semicircular canal stimulated. Any pair of semicircular canals can be stimulated by rotation with the head in a suitable position. Destruction of one labyrinth causes rhythmic nystagmus towards the opposite side, which ceases if the other labyrinth is destroyed.

Miners' Nystagmus occurs chiefly in those who have worked long at the coal face. The patient complains of defective vision which is worse at night, headache, giddiness, photophobia, the dancing of lights and movements of objects. The nystagmus is essentially rotatory and very rapid; in latent cases it is elicited by fixing the head and making the patient look up. In severe cases the lids are nearly closed and the head is held backwards; there is tremor of the head and eyebrows. The frequency of the disease varies inversely with the illumination in the mine suggesting that fixation difficulties in the dim illumination may be an ætiological factor; it will be remembered that vision in a dull light is carried out almost entirely by the rods. In these circumstances visual acuity is greatest 10° – 15° outside the fovea, and there is a physiological central scotoma making fixation difficult. There is, however, a large psychoneurotic factor in all cases. Improvement in miners' lamps and in the lighting of mines eliminates the disease which is a cause of considerable economic loss in time and compensation.

The prognosis is good in spasmus nutans, and in miners' nystagmus if the occupation is changed, although recovery is slow. In all other cases it is bad, but nystagmus tends to diminish with advancing years. Treatment is therefore palliative—the correction of refraction, the use of smoked glasses in albinism, and the treatment of any disease which may be present.

CHAPTER 30

CONCOMITANT STRABISMUS : HETEROPHORIA

WE have already seen that in concomitant squint, as opposed to the paralytic type, although the eyes are misaligned, they retain their abnormal relation to each other in all movements. In paralytic squint the afferent pathways and centres are intact, but the efferent, effector mechanism breaks down. In concomitant squint the efferent pathways are normal and can still maintain co-ordination of the eyes, but either the afferent path is defective (usually due to poor visual acuity owing to a defect in the eye) or the central mechanism mediating the fixation and fusional reflexes is undeveloped or has broken down. The break-down may be due to peripheral causes, such as the excessive effort of convergence required with the sustained accommodation necessary in hypermetropes or a slight weakness in an extra-ocular muscle such as is not sufficient to cause a paralytic squint.

If the fusion mechanism is well-developed and the defect slight, visual alignment may be maintained in normal circumstances by a continued effort of fusion: the squint is then *latent* and can only be made manifest when fusion is made impossible (as by covering up one eye). This condition is called *heterophoria* or *latent squint*. If, on the other hand, the maintenance of alignment becomes impossible, a true or *apparent concomitant squint* develops and remains constant.

HETEROPHORIA OR LATENT STRABISMUS

We have just defined heterophoria as a condition wherein there is a tendency to misalignment of the visual axes which is corrected by the fusional capacity. As with all deviations the tendency is equally shared between the two eyes. Since the position of rest is usually one of slight divergence, few people are *orthophoric* and some degree of heterophoria is almost universal. If the latent deviation is one of convergence the condition is called *esophoria*, of divergence, *exophoria*, if vertical, *hyperphoria*: it is impossible to be sure whether there is absolute hyperphoria of one eye or hypophoria of the other, the condition being relative. If the deviation is torsional the term *cyclophoria* is applied. Lateral deviations are the most common, due often to over-stimulation of convergence with accommodation in hypermetropia (*esophoria*) or under-stimulation in myopia (*exophoria*). Hyperphoria is also common and is probably often due to abnormal insertions or slight

weakness of one or other of the vertically rotating muscles. Cyclophoria is rare.

The *symptoms* of heterophoria may be considerable since parallelism of the visual axes is only maintained by tonic contraction of the appropriate muscles. Symptoms of eyestrain are therefore encountered in the higher degrees ; but the smaller degrees give rise to little or no trouble. This particularly applies to eso- and exophoria since the muscles involved are accustomed to act unequally in convergence ; only when the deviation is great— 5° to 10° or more—is asthenopia generally present. Slight degrees of hyperphoria, however, may cause discomfort, for in these cases more complicated adjustments are necessary involving the non-physiological action of muscles which are not accustomed to work together in order to keep the visual axes in the same plane. For the same reason cyclophoria gives the greatest discomfort of all.

As might be expected the deviation is liable to become manifest in conditions of bodily fatigue and to vary in amount from time to time. Some periodic squints are due to this cause, and the periodicity may be rhythmic. Thus a child may squint in the evening when he is tired ; after a good night's rest the squint may disappear and may not return until the second or third day, the sequence being accurately repeated. Often latent squints give no trouble until school age arrives or adult life is reached when the demands of near vision increase the strain. No symptoms arise perhaps until after reading or writing for an hour or two when "the letters seem to run together." This is due to relaxation of the over-strained muscles ; the eyes momentarily assume the position of rest, and diplopia, which is not appreciated as actual double vision, causes blurring of the print. With an effort the blurring is overcome, but eventually this becomes impossible, headache supervenes, and the work has to be abandoned.

The *diagnosis* of heterophoria simply depends on abolishing fusion so that, without its control, the eyes assume their position of rest. Several tests for this are available.

The most simple is the *screen test* (p. 474). When a distant object is regarded and both eyes are uncovered there is no deviation. One eye is then screened and the latent deviation appears ; when the screen is removed this eye moves at once to regain the position of binocular fixation. The other eye reacts similarly, the deviation in both being the same.

Other tests depend on altering the appearance of the retinal image in one eye so that no stimulus is given to fusion.

Of these the simplest is the *Maddox Rod Test*. The patient is placed six metres from a bright spot of light in a dark room. A Maddox rod, which consists of four or five cylinders of red glass side by side in a supporting disc, is placed in the trial frame before one eye ; the same effect is given by a

disc of deeply grooved red glass (the Maddox groove, Fig. 388). The spot of light seen through the red cylinders appears as a long red line. If the cylinders are placed with their axes horizontal the red line will be vertical. If there is orthophoria the bright spot will appear to be in the centre of the vertical red



FIG. 388. Maddox groove.

line ; if there is eso- or exophoria the red line will be to one side of the spot. The angle of the deviation is measured by the strength of the prism which it is necessary to place in front of the Maddox rod (or the other eye) in order to bring the red line and the spot together. The nature of the deviation is indicated by the position of the base of the prism, whether out or in. The Maddox rod is then rotated so that the cylinders are vertical ; the red line will now be horizontal. If there is no hyperphoria the line will pass through the bright spot. If there is hyperphoria the red line will be below or above the spot according to whether the relative hyperphoria is associated with the eye with the rod in front of it or with the other. In each case the

amount of deviation is measured either on a graduated tangent scale set on the wall (Fig. 389) or by the strength of the prism required to correct it.

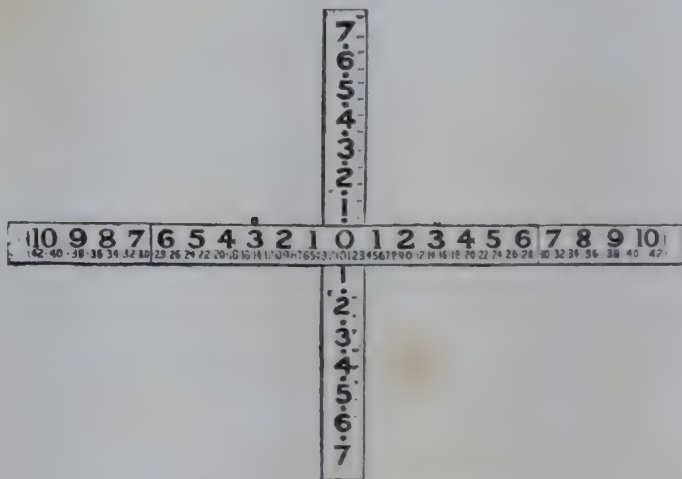


FIG. 389. Tangent scale.

The deviation in latent squint is often different in near vision from that in distant vision. An exophoria appearing when near objects are regarded is in fact an *insufficiency of convergence*, a condition which may give rise to symptoms when much near work is undertaken.

The deviation in near vision is conveniently tested by the *Maddox wing test* (Fig. 390) in which, when the patient looks through the two slit-holes in the eyepieces of the instrument, the fields which are exposed to each eye are separated by a diaphragm in such a way that they glide tangentially into each other. The right eye sees a white finger pointing vertically upwards and a red arrow pointing horizontally to the left. The left eye sees a horizontal row of figures in white and a vertical row in red. These are calibrated to read in degrees of deviation. The finger pointing to the horizontal row of figures

and the arrow pointing to the vertical row should both be at zero ; any deviation indicates an eso- or exophoria or a hyperphoria, the amount of which can be read off on the scale. There are several *diaphragm tests* based on somewhat similar principles.

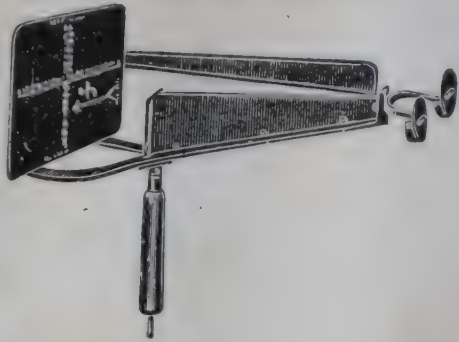


FIG. 390. The Maddox wing test.

Besides the actual measurement of the deviation in latent strabismus the strength of the muscles involved should also be tested by forcing them to a maximum effort against prisms (*prism vergence tests*). With the patient seated six metres from a light the highest prism which can permit single vision gives the *verging power* for the particular direction involved. The converging power varies very much and with practice can be raised to the neighbourhood of 50 degrees (25° d) or more ; if it falls below 20 degrees, it may be taken to be definitely insufficient. The diverging power should be 4 to 5 degrees, and the normal limits of super- and subduction are from 1.5 to 2.5 degrees.

Treatment. The lower degrees of esophoria, and to a less extent of exophoria, cause no symptoms and need no special treatment. If symptoms are apparent after any error of refraction has been corrected with spectacles, a rational treatment of eso- or exophoria consists in exercising the weak muscles against prisms, or by the use of the stereoscope. Unfortunately this is usually not or only temporarily beneficial, but relief may be maintained by repeating the exercises at intervals, an activity which the patient can practise himself. If this is ineffective the symptoms may be relieved by ordering prisms in spectacles to correct the defect, i.e., prisms with their bases directed in the opposite sense to those used for exercise. This should be avoided if possible since it generally tends to increase the defect, so that stronger prisms have to be ordered from time to time. The total prismatic error should be divided equally between the two eyes in ordering the correction. Hyperphoria in its less degrees should unhesitatingly be treated in this way: in this condition exercises are useless.

In all cases when the deviation is large and is unaffected by such treatment, operation may be considered, one or other muscle being recessed or resected, the case being treated as if it were a manifest squint (*q.v.*). Even if not curative, the deviation may thus be reduced to a degree which is controllable by prisms.

Insufficiency of convergence may be treated by prism exercises. The following simple exercise is often sufficient without having recourse to prisms. A pencil is held in the hand and slowly approximated to the nose until, despite an effort of convergence, it appears double ; this is repeated until the distance at which diplopia

occurs is gradually shortened. At about every tenth time the patient looks into the distance, so as to relax his accommodation and convergence. The exercise should be repeated three or four times a day for several weeks. More effective are orthoptic exercises with stereoscopic apparatus. If convergence training fails, prisms, base in, may be ordered with the reading lenses. In these cases care must be taken not to over-correct presbyopia (p. 79).

CONCOMITANT STRABISMUS

We have already seen that in concomitant strabismus, the visual axes, although abnormally directed, retain their abnormal relation to each other in all movements of the eyes. The final lower pathway in the efferent tracts controlling ocular movements is undisturbed, but either the visual impressions arriving at the cortex from one eye are defective or the fusional reflexes have not developed or have been weakly developed and have broken down so that an ocular deviation results. The cause or causes of this failure are unknown and many theories have been stated and restated so frequently that they are often accepted as proved. The fact remains, however, that no theory of the fundamental causation has yet been advanced which satisfactorily explains the condition.

Many important factors in the ætiology of concomitant strabismus are known, and a proper appreciation of them is essential to rational treatment. In the first place, defective vision in one eye, such as high ametropia, opacities in the media or ocular disease, makes it easy for the affected eye to lose fixation. If the defect exists from birth or early life, the cortical cells normally subserving both eyes may never develop binocular connections so that fusion is impossible. Disturbances in muscular equilibrium usually due to a congenital mal-insertion or defective development of one or more of the extrinsic muscles, may act in the same way, the squint being perhaps preceded by a period of heterophoria during which fusion was maintained. The forced dissociation between accommodation and convergence, a matter early pointed out by Donders, is also of importance; the continuous effort of accommodation in the hypermetrope in order to see clearly even in the distance stimulates convergence to a greater degree than is compatible with binocular fixation; faced with the dilemma of either relaxing his accommodation and not seeing clearly or converging too much, he chooses the latter and squints inwards. Conversely, the myope squints outwards. These relationships between the refractive condition and the direction of squint, however, are by no means invariable.

Concomitant strabismus may be either convergent or divergent.

Convergent strabismus is the more common and is most frequent in hypermetropes. It always commences in childhood. It may become manifest after a fright, an attack of whooping cough, measles or other debilitating illness, and is often popularly

attributed to some such cause. The deviation is not always quite horizontal: in many cases the eye deviates upwards as well as inwards. In most of these cases there is a congenital element, and the deviation may have been primarily paretic.

Divergent strabismus, on the other hand, is most common in myopes, often commencing at a later age; it may, indeed, arise late in life when one eye loses most or all of its vision. The better eye is then used and the other is allowed to take up the position of rest, which is usually one of divergence. There is an undoubted tendency for the deviation in all cases of convergent strabismus to diminish with the diminution of accommodation in age. Spontaneous cure rarely if ever occurs in divergent strabismus, which tends to increase with time.

If one eye habitually fixes and the other squints the case is usually termed one of *unilateral strabismus*. Sometimes fixation is retained by either eye in which case the squint is said to be *alternating*. Usually in a divergent squint an object towards the right in the field of vision will be fixed with the right eye, in the left of the field by the left eye; the converse may occur in convergent squint. Occasionally patients with alternating strabismus can fix with either eye voluntarily, but usually they are unconscious of which eye is fixing. Concomitant squint may be *constant*, or occur only at intervals—*periodic*.

The Investigation of Strabismus. The first step is to ensure that any apparent deviation is indeed real. An *apparent squint* may be due to the configuration of the palpebral aperture. If, for example, as commonly occurs in children, epicanthus (*q.v.*) is present and the medial canthi approach the cornea, the appearance of an internal squint results. More commonly such an appearance is due to a divergence between the visual axis and the optic axis, a divergence which gives the appearance of convergent squint to myopic eyes, of divergent squint to hypermetropic eyes—the opposite relation to a real squint.

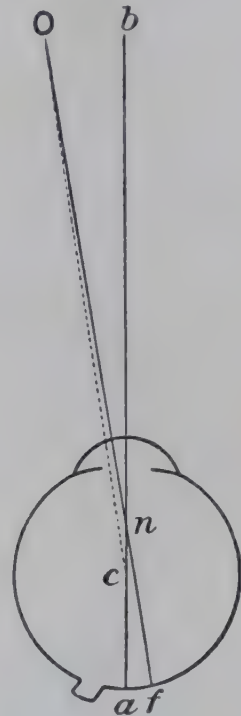


FIG. 391. Apparent strabismus. *a b*, optic axis upon which the refractive surfaces are centred; *f*, fovea centralis; *n*, nodal point; *c*, centre of rotation; *O*, point of fixation; *O c*, line of fixation; *O n f*, line of vision; *O c b*, angle γ . It is practically equal to *O n b*, which can be measured. In actual practice the guide to *a b* is taken from the centre of the pupil; *a b* does not usually pass accurately through the centre of the pupil, so that the result is always only approximate. The angle γ is to the nasal side in hypermetropia and emmetropia.

The optic axis upon which the cornea and lens are centred passes through the centre of rotation of the eye and approximately through the centre of the pupil. The visual axis passes through the nodal point and the fovea centralis, thus crossing the optic axis and making a small angle with it. This angle (although the convention is slightly inaccurate) is commonly spoken of clinically as the angle γ (gamma) (Fig. 391). In the emmetropic eye the angle γ is said to be positive, since the optic axis usually cuts the retina internal to the fovea centralis. In hypermetropic eyes the angle γ is also positive but greater than in emmetropia. In myopia the angle γ is absent or negative for the visual axis and the optic axis coincide or the latter cuts the retina external to the fovea centralis.

Neither of these lines can be seen, and the direction of the line of vision is judged by the position of the pupil. Hence the greater the size of a positive angle γ the more the eye will appear to look outwards. If the angle γ is negative the eye will appear to look inwards. In high hypermetropia, therefore, there will be an apparent divergent squint, in high myopia an apparent convergent squint. The latter is the more striking because the emmetropic eye usually has a positive angle γ of 5° , thus producing an apparent divergence of 10° , which, however, we are accustomed to regard as the normal position of the eyes.

The next step is to differentiate a concomitant from a paralytic squint. Both these conditions are analysed by the *screen test*. In an apparent squint there is no deviation when either eye is covered up and then is uncovered to resume fixation. In paralytic squint we have already seen that the secondary deviation is greater than the primary; in concomitant squint, both deviations are equal. No extra effort is required to look in any direction, so that when either eye is covered and then uncovered, the deviation suffered by each is the same. If, moreover, the movements of the eye are found to be full in all directions, and there is no complaint of diplopia, it may be concluded that there is no paralysis. It must be remembered, however, in performing this test in a marked squint, that the eyes do not move as much as usual in the direction opposite to that of the deviation. Thus, in convergent squint it may be very difficult to get the eyes to move outwards to the full extent so that the margin of the cornea lies inside the lateral canthus. This defective movement may be due to muscular contracture.

When the fixing eye is covered with the screen the deviating eye usually moves so as to take up fixation. In unilateral squints of long standing this eye may remain motionless or move only slightly, a condition which is called *eccentric fixation*. Since it occurs only with marked deviation of long standing there is generally no difficulty in distinguishing it from apparent squint.

Apart from the loss of binocular vision, concomitant squint is asymptomatic: diplopia may be present in the initial stages, but it rapidly disappears due to psychological suppression of the macular image of the squinting eye. In most cases suppression is aided by an actual visual defect in this eye, but it also occurs in alternating squint, in which both eyes are frequently normal or have the same degree of ametropia. Suppression is doubtless aided in all

cases by the peripheral situation of the image in the squinting eye, but the essential seat of suppression is in the brain. Since the image of any object falls on disparate points diplopia results, and since the brain finds this intolerable, it actively inhibits the image of the squinting eye. This prolonged active suppression results in a permanent lowering of the vision of the squinting eye—*amblyopia ex anopsia*. In contrast, it is noteworthy that, because this purposeful and active inhibition is not involved, an eye which has been blind for many years from cataract, even of the congenital type, immediately attains good vision after a successful operation.

The suppression affects mainly the fovea ; indeed, the acuity of vision may become greater at the eccentric point of the retina where

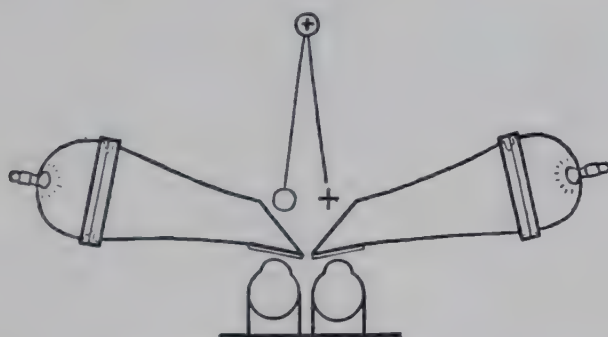


FIG. 392. The Amblyoscope.

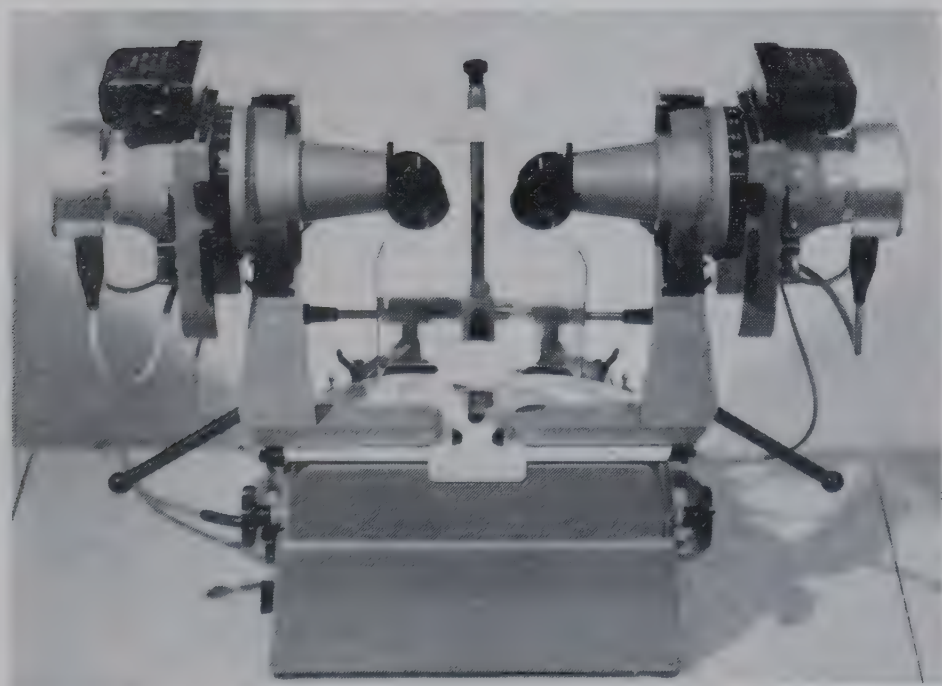


FIG. 393. A modern synoptophore for examination of binocular vision, the capacity for fusion and the muscle balance (Clement Clarke).

the new fixation axis falls habitually in the squinting position, a result which leads to the development of a "false macula" with abnormal retinal correspondence and abnormal projection. This abnormal relationship is maintained only when both eyes are in use; when the squinting eye only is used, the fovea is usually (but not invariably) used again for fixation. So fixed may this abnormal system become that if the eyes are straightened, diplopia may result, to overcome which the eyes naturally tend to return to their old squinting position. The elimination of false correspondences is therefore of importance before operation is attempted. Eventually all power of fixation may be lost by the amblyopic eye.

Measurement of the angle of the deviation is important in all cases of concomitant squint as a guide to treatment. Many methods are available, but much the most useful and accurate is by a calibrated *amblyoscope* (the *synoptophore* or *orthoptoscope*, an instrument based on the principle of a simple amblyoscope) (Figs. 392-3). The patient looks down two adjustable tubes at two easily fixated small objects, and the angle between the tubes is altered until each eye attains fixation when they are used separately, one rapidly after the other. This gives the position of the visual axes. The corneal reflexes are then centred on the pupil; this gives the position of the optic axes. The difference between the two gives the angle of deviation. An attempt is then made to make both eyes fixate the objects simultaneously so that they appear superimposed; if the deviation is different from that obtained with each eye separately, the presence of abnormal correspondence is shown, and the difference gives the angle of false projection. Other less accurate methods are available.

A rough indication of the angle of the squint can be obtained from the position of the corneal reflex when light is thrown into the eye with the ophthalmoscope, from a distance of about two feet (Fig. 394). The patient is told to look at the light; an infant does this reflexly. In the fixing eye the corneal reflex will be in the centre of the pupil, or slightly to the inner side if there is a large angle γ , to the outer side if there is a negative angle γ . The light is then turned onto the squinting eye. If the reflex is about halfway between the centre of the pupil and the corneal margin there is a deviation of about 20° ; if it is at the corneal margin, about 45° . This test gives only an approximate estimation.

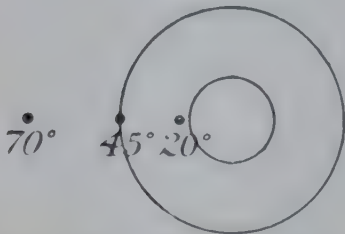


FIG. 394. Diagram of the position of the corneal reflex as a guide to the angle of the squint.

The angle of deviation of the squinting eye can also be measured on the perimeter or the tangent scale; in either case the patient fixes the central point with the good eye, and the surgeon carries a light along the arc of the perimeter or the arm of the tangent scale until the corneal reflex thus obtained is centred on the pupil of the squinting eye. The angle at which this occurs is the angle of squint (Figs. 395-6).

Treatment. The routine treatment of a case of concomitant convergent strabismus in a child is as follows:—

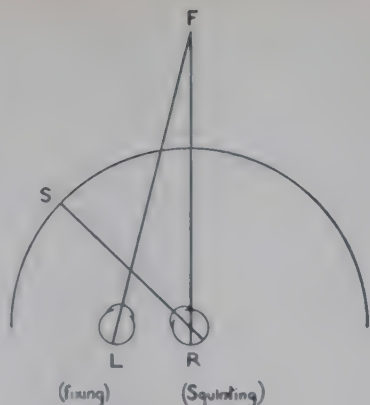


FIG. 395.

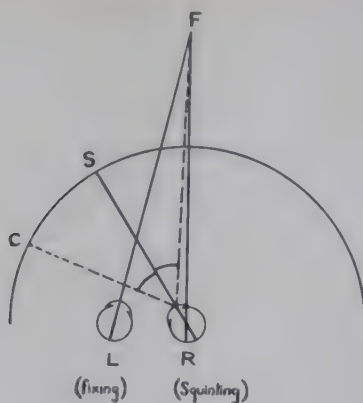


FIG. 396.

FIGS. 395-6. Angular strabismometry. Fig. 395, Javal's method. The R.E. (squinting) is central in the perimeter. The L.E. fixes F. The surgeon carries a light (S) along the arc of the perimeter until the corneal reflex in R is central. Angle SRF (less the angle γ) is the angle of squint. Fig. 396, Charpentier's method. The R.E. (squinting) is central in the perimeter. The L.E. fixes F. A light is placed on the perimetric arc on the line FR and the surgeon moves round the arc (to C) until he sees the corneal reflex in R central. Half the angle FRC (i.e., FRS) (less the angle γ) is the angle of squint.

(1) *Preliminary.* Record the distant vision of each eye if the child is not too young (by the E Test, p. 133, if necessary), the angle of deviation, and any false projection. Order ung. atropinæ, 1 per cent., three times a day for at least four days. At the end of this period estimate the error of refraction by retinoscopy and confirm the result subjectively if possible; reliance should be placed on the retinoscopy rather than on subjective tests. Again measure the angle of the squint, which is likely to be less under atropine than without a mydriatic. Order the full spectacle correction for constant use. A smaller deduction for the effect of atropine should be made than in hypermetropia without squint; indeed, if the refractive error is small no deduction may be made. Great care must be taken to correct all astigmatism, especially in the squinting eye. The patient should be re-examined in a month's time.

If the child is less than two years old, is a hypermetrope and has an internal squint, it is easier to eliminate accommodation by keeping both eyes under the influence of atropine; the 1 per cent. ointment need be applied only once every second day. The child should be examined at regular intervals until it is considered advisable to order spectacles.

(2) *Occlusion of the Fixing Eye.* If, when the vision is tested, the squinting eye is amblyopic, as is usually the case, an effort should be made to improve the vision in it by continual exercise. In order that this eye may be used, the other must be prevented

from seeing, or at any rate from seeing clearly. The only satisfactory method of ensuring this is by complete occlusion, effected by a patch covering the better eye; to prevent the child removing it, it is well to fix it to the skin by adhesive material. The patch is changed when it becomes dirty or loose. *Occlusion should be absolute* since if both eyes are used together active inhibition of the squinting eye rapidly undoes any good result achieved; it may have to be continued for six to twelve weeks, but if there is little improvement after this interval the practice may be discontinued. In cases of eccentric fixation the good eye should be occluded for a time in the hope that foveal fixation will develop in the other. In some cases the deviation becomes transferred to the occluded eye: this is a good sign, as it indicates that the vision with the originally squinting eye is only slightly worse than that of the fixing eye.

In very young children or in recent squinters in whom the habit of suppression has not become fixed, the less drastic procedure of instilling atropine into the *fixing* eye every two days may be sufficient; in this event the squinting eye must be used for seeing near objects.

(3) *Orthoptic Training*. The further treatment depends upon the size of the angle of deviation, the condition of vision in the squinting eye, and a variety of other factors which differ in each case. An attempt is made to cultivate binocular vision and stereoscopic fusion by *orthoptic training*. This consists essentially in specially designed exercises undertaken mainly on an orthoptoscope devised, not to increase the power of the muscles (which is unimpaired) but to encourage the development of binocular vision and the capacity for fusion. It is obvious that only a small and recently acquired squint can be cured in this way and in the majority of cases such training must be combined with surgery. As a rule in this way only can a complete "cure" be attained since the patient is placed in the same condition as a normal person; his eyes are straight and he has binocular vision. The eyes can be put straight surgically, but this cures only the deviation and leaves the fundamental disability unaltered. If eccentric fixation is well established, it is often well to occlude the affected eye for some weeks and then to stimulate the macula by special *pleoptic methods* (flashing devices, the production of after-images, etc.); but such treatment is time-consuming and exacting and not always successful.

There are three stages in orthoptic treatment: (a) the production of simultaneous vision with the two eyes; (b) the production of binocular vision and the elimination of false projection; and (c) the production of stereoscopic vision, i.e., the fusion of two images of the same object seen in perspective, resulting in the perception of solidity and relief.

Although orthoptic treatment is of great value in inculcating correct visual habits such as the training of binocular vision and the

abolition of false projection, it rarely if ever by itself cures a squint of over 10° deviation or one of long standing. In many cases it is useless to attempt it, and in all cases it is useless unless carried out systematically and thoroughly, and it is rarely worth while to persist with it as a sole method of treatment if the deviation is not corrected within three months. For the details of the treatment monographs on the subject must be consulted. Its most valuable function is as an adjunct to operative treatment—to develop binocular vision and reduce the intensity of abnormal projection before, to consolidate results and correct any residual deviation after surgery.

(4) *Surgical treatment* of concomitant unilateral convergent squint is indicated when the angle of squint is 10° or more when wearing correcting lenses, and in children when orthoptic training has failed to correct the deviation within a reasonable time. As a general rule it should be undertaken early and certainly as soon as the child is old enough to co-operate in post-operative orthoptic treatment, usually between four and five years of age. Postponement until the child is ten or more usually results in the permanence of amblyopia and failure to establish binocular vision. The operation is then purely cosmetic.

The safest operations for general use are a resection or a recession of the appropriate muscle. Free tenotomy of the medial rectus tendon and its expansions into Tenon's capsule is usually followed by divergence and retraction of the caruncle and plica semilunaris, owing to failure of reattachment to the globe. A tenotomy of the lateral rectus muscle, however, is sometimes permissible. The medial rectus should not be recessed more than 5 mm. lest weak convergence occur, leading to discomfort in reading and near work and to headaches. A medial rectus recession of 5 mm. will correct about 12° of squint. Considerable experience is required to assess the amount of recession and advancement needed in different cases, but in general one mm. of displacement of the insertion of a muscle corrects 2.5° of deviation.

If the deviation is 10° or less, a recession of the medial rectus of the squinting eye usually suffices.

If the deviation is more than 10° , resection with or without advancement of the lateral rectus of the squinting eye, usually with recession of the medial rectus of the same eye, will be necessary. If the deviation is large, similar procedures should be carried out on the other eye, deferred, if convenient, to a later date when the degree of residual deviation can be accurately assessed.

The treatment of alternating concomitant convergent squint without appreciable error of refraction is purely cosmetic. These patients have no binocular vision, and it is useless to attempt to develop it unless the case is seen when the patient is very young or immediately after the squint has been first noticed. There is often a considerable deviation so that surgery may be required on both eyes.

The treatment of concomitant divergent strabismus is similar to that of the convergent type. The refraction must be first carefully corrected, and it is advisable to order a full correction for constant use unless the myopia is very high. Recession of the lateral rectus is seldom indicated in these cases because the benefit derived is too slight; it will not correct much more than 5° deviation. Hence resection of the medial rectus is usually necessary, reinforced, if required, by recession of the lateral muscle. In divergent strabismus slight over-correction is indicated, for these eyes show a great tendency to revert to their former position.

Sometimes the patients develop diplopia after the eyes have been straightened. This may be due to false projection (p. 455), but also occurs with alternating squints. It is a troublesome complication, usually persisting for some weeks or months, and is distressing to the patient; but it usually eventually disappears.

OPERATIONS ON THE EXTRINSIC MUSCLES

In operating for squint with a general anæsthetic it is important to remember that the position of the eyes varies in different stages of anæsthesia so that it gives no criterion of the final position after the anæsthetic has passed off; in squint surgery the deviation as previously measured should be remembered and the position actually present under the anæsthetic ignored. With local anæsthesia discomfort results when tension is put upon a muscle—which is almost impossible to avoid. In the case of children a general anæsthetic should be used and any small residual deformity dealt with by orthoptic exercises. In all cases the preparation of the eye for operation should follow the lines already indicated, as also should post-operative dressings (p. 405). Catgut (0000) will save a second anæsthetic for the removal of the stitches, and eyeless needles should be used.

Recession of the Medial Rectus. A vertical incision is made with scissors in the conjunctiva over the medial rectus and a flap undermined towards the inner canthus exposing the muscle covered by Tenon's capsule. Tenon's capsule is then button-holed with scissors and slit for 7 mm. along the upper and lower edges of the muscle; the part of the capsule covering the muscle should be preserved. The point of a strabismus hook (Fig. 326) is passed into Tenon's capsule at the posterior limits of the incisions and retracted. Dividers measuring the amount desired to set the muscle back are placed along the upper and lower borders of the muscle, the distance measured off from the tendon insertion, and marked on the sclera. Catgut sutures are passed through the upper and lower edges of the muscle 2 mm. behind its insertion in the so-called "whip stitch" fashion (Fig. 397). The tendon is divided, and the stitches are passed through the superficial layers of the sclera at right angles to the long axis of the muscle at the points already marked. They are tied, and the conjunctival incision is then closed with a continuous suture.

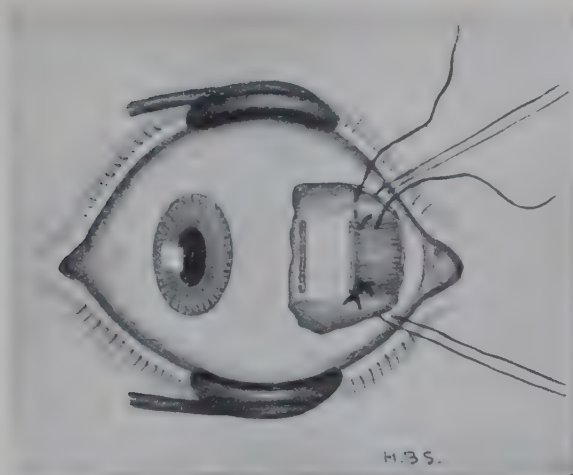


FIG. 397. Recession of medial rectus.

Resection of the Lateral Rectus. An incision is made 2 mm. behind and concentric with the corneo-scleral junction in front of the insertion of the muscle, and the conjunctiva undermined. The muscle is exposed in the same manner as in recession. A strabismus hook is passed between the muscle and sclera, and the length of muscle and tendon for resection determined and marked with gentian violet.

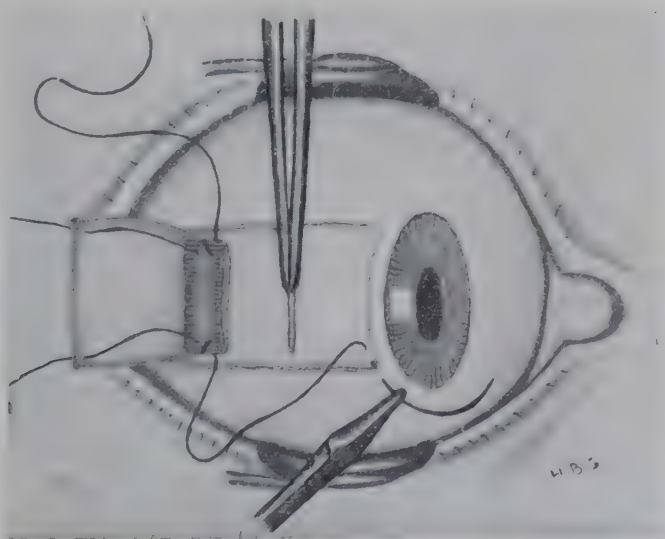


FIG. 398. Advancement and resection of lateral rectus.

Whip-stitch sutures are passed through the upper and lower edges of the muscle 2 mm. behind the mark and ensnaring a breadth of about 2 mm. of the muscle fibres. The muscle is divided at the mark, the distal part being held in fixation forceps so as to steady the globe whilst the scleral sutures are inserted. The needles are passed through half the thickness of the sclera transversely to the long axis of its fibres at the anterior marked spots (Fig. 398). The tendon is then divided at its

insertion, the shortened muscle drawn forwards, the sutures tied, and the conjunctival incision closed. If no more than 6 to 8 mm. of shortening of the muscle is required, this amount may be excised and the muscle stitched to its original insertion (*resection*). For larger corrections the necessary amount is more easily made up by stitching the resected muscle in front of its insertion (*advancement*).

The superior and inferior recti are resected or advanced and recessed in the same way.

A simple tenotomy can only be safely performed upon the lateral rectus, and even then is better replaced by a recession. The result which can be expected is only a correction of 5° .

A myectomy of the inferior oblique is done after approaching the muscle near its origin from the inner wall and floor of the orbit or, more easily, near its insertion beneath the lateral rectus. In the former case the incision may be through the skin of the lower lid or the conjunctiva of the lower fornix; in the latter case a horizontal incision is made through the bulbar conjunctiva and Tenon's capsule at the level of the lower border of the lateral rectus. In either case the muscle is drawn out by a strabismus hook and a length of 2 to 3 mm. excised, the muscle being first crushed with artery forceps to forestall hæmorrhage.

The techniques for advancements and recessions of the superior and inferior obliques are outside the scope of this book.

SECTION VI

DISEASES OF THE ADNEXA OF THE EYE

CHAPTER 31

DISEASES OF THE LIDS

Anatomy. The lids are covered anteriorly by skin and posteriorly by mucous membrane—conjunctiva tarsi. They contain muscle, glands, blood vessels and nerves, all bound together by connective tissue which is particularly dense at the posterior part where it forms a stiff plate—the tarsus (Fig. 399).

The skin of the lids is peculiar in its thinness, its loose attachment, and the absence of fat in its corium. It is covered with fine downy hairs, which are provided with small sebaceous glands, and there are also small sweat glands. At the margins these structures are specially differentiated. The cilia or eyelashes are strong, short, curved hairs, arranged in two or more closely set rows. Their sebaceous follicles, like the cilia themselves, are specially differentiated, and are called *Zeis's glands* which, apart from being larger, are identical with other sebaceous glands. The sweat glands near the edge are also unusually large and are known as *Moll's glands*; they are situated immediately behind the hair follicles, and their ducts open into the ducts of Zeis's glands or into the hair follicles, not directly onto the surface of the skin as elsewhere.

The margin or free edge of the lid is called the *intermarginal strip* (Fig. 400). It is covered with stratified epithelium which forms a transition between the skin and the conjunctiva. The anterior border is rounded; the posterior, which lies in contact with the globe, is sharp. The capillarity induced by this sharp angle of contact is of importance in the proper moistening of the surface of the eye. Immediately anterior to the posterior border the orifices of the ducts of the meibomian glands form a single row of minute orifices, just visible to the naked eye. Between them and the anterior border is a fine grey line, which is important in operations in which the lid is split since it indicates the position of the loose fibrous tissue between the orbicularis palpebrarum and the tarsus.

The tarsus consists of dense fibrous tissue; it contains no cartilage. Embedded in it are some enormously developed sebaceous glands, the *meibomian (tarsal) glands*, consisting of nearly straight tubes, directed vertically, twenty to thirty in number in each lid, each opening by a single duct on the margin of the lid.

The orbicularis palpebrarum occupies the space between the

tarsus and the skin. The main central band of the levator palpebræ superioris is inserted into the upper border of the tarsus; an anterior slip passes between the bundles of the orbicularis to be inserted into the skin of the middle of the lid; a posterior slip is inserted into the conjunctiva at the fornix. The inferior rectus and oblique muscles send fibrous strands forwards into the lower lid to be attached to the tarsus and palpebral ligament.



FIG. 399. Section through the upper lid. 1, orbicularis muscle; 2, sweat gland; 3, hair follicle; 4, gland of Zeis; 5, cilium; 6, gland of Moll; 7, pars marginalis of orbicularis muscle; 8, pars subtarsalis of orbicularis muscle; 9, inferior arterial arcade; 10, meibomian gland; 11, gland of Wolfring; 12, conjunctival crypts; 13, superior arterial arcade; 14, gland of Krause; 15, Müller's muscle; 16, levator palpebræ superioris; 17, fat.

Besides these striped muscles there are layers of unstriped muscle in each lid, constituting the superior and inferior tarsal muscles of Müller. The fibres of the former arise among the striped fibres of the levator, pass down behind it, and are inserted into the upper border of the tarsus; the inferior tarsal muscle lies below the inferior rectus and is inserted into the lower tarsus.

The arteries of the upper lid form two main arches, the superior lying between the upper border of the tarsus and the orbicularis, the inferior, in a similar position just above the hair follicles. In the lower lid there is one arch near the free edge. There are two venous plexuses in each lid: a post-tarsal passing into the ophthalmic veins, and a pre-tarsal opening into subcutaneous veins.

The sensory nerve supply is derived from the trigeminal. The third nerve supplies the levator palpebræ, the seventh the orbicularis, and the sympathetic Müller's muscles.

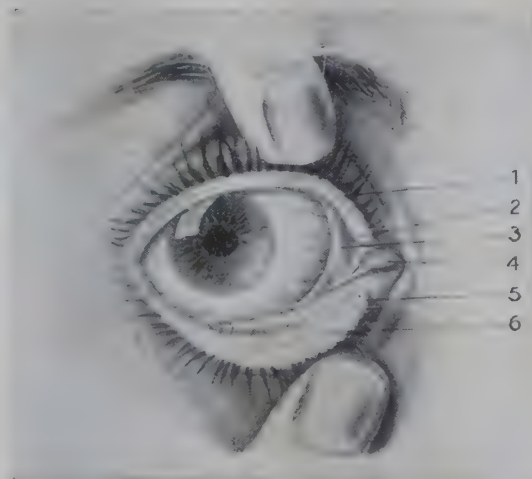


FIG. 400. The surface anatomy of the lid margins and the conjunctival sac. The lids have been pulled aside to display their margins and expose the structures at the inner canthus. 1, intermarginal strip ; 2, upper punctum ; 3, plica semilunaris ; 4, caruncle ; 5, lower punctum ; 6, openings of tarsal glands.

Œdema of the lids is common and, owing to the looseness of the tissue, may be so great as to close the eye (Fig. 401). It may be inflammatory or passive. *Inflammatory œdema* may be caused by an inflammation of the lid itself (dermatitis, stye, insect bite, etc.), of the conjunctiva (when it may be associated with chemosis), of the lacrimal sac, or by purulent inflammation in the eye, Tenon's capsule, the orbit or the underlying nasal sinuses. Chronic thickening of the lids, resembling œdema but harder in consistency, may follow recurrent attacks of erysipelas—so-called *solid œdema*.

Passive œdema, due to circulatory obstruction, is seen in general disease (renal disease, cardiac failure) or local conditions such as cavernous sinus thrombosis. An intermittent and acute œdematous condition due to unstable vasomotor reactions, frequently deter-



FIG. 401. Œdema of the lids ; an allergic case (Hollander).

mined on an allergic basis, is relatively common in the lids (angio-neurotic œdema).

INFLAMMATION OF THE LIDS

Almost any of the inflammatory conditions which affect the *skin* in general may attack the lids. In this region dermatitis is common and frequently marked, particularly allergic manifestations due to sensitization to innumerable allergens—cosmetics, dyes, drugs, etc. Atropine irritation is a typical example. An eczema may occur in association with a discharging conjunctivitis or where there is much lacrimation. The ordinary coccal infections cause boils and abscesses ; specific infections may occur such as anthrax or zoster (*q.v.*). Erysipelas is dangerous in that it may spread to the orbit, leading to cellulitis and atrophy of the optic nerve, thrombosis of the cavernous sinus, or meningitis.

Blepharitis is a chronic inflammation of the margins of the lids, appearing as a simple hyperæmia or as a true inflammation which may occur in two forms. In *squamous blepharitis* small white scales accumulate among the lashes which fall out readily, but are replaced without distortion (Plate XXI, Fig. 1). If the scales are removed the underlying surface is found to be hyperæmic, but not ulcerated. The condition is often essentially metabolic and of the nature of seborrhœa associated frequently with dandruff of the scalp. Such ætiological factors require treatment.

Ulcerative blepharitis is an infective condition. Yellow crusts glue the lashes together and on removing them small ulcers which bleed easily are seen around the bases of the lashes. This distinguishes the condition from matting together of the lashes by conjunctival discharge ; removal of these crusts reveals normal lid margins. The symptoms are redness of the edges of the lids, itching, soreness, lacrimation, and photophobia (Plate XXI, Fig. 2).

The sequelæ of the ulcerative form are serious. If not treated energetically and with perseverance the disease is extremely chronic, causing or being accompanied by chronic conjunctivitis. The ulceration is liable to extend deeply, destroying the hair follicles so that the lashes fall out and either are not replaced or only by a few small, scattered, distorted cilia (*madarosis*). When the ulcers heal the cicatricial tissue contracts. Neighbouring hair follicles are drawn out of place and a false direction is given to the remaining cilia so that they may rub against the cornea (*trichiasis*). Occasionally the development of cicatricial tissue may be extreme so that the edge of the lid becomes hypertrophied and droops in consequence of its weight (*tylosis*).

The lower lid is particularly liable to be displaced by prolonged ulcerative blepharitis. The contraction of the scar tissue drags the conjunctiva over the margin ; the posterior lip of the intermarginal strip, instead of being acute-angled, becomes rounded, so that its

PLATE XXI
INFLAMMATIONS OF THE LIDS



FIG. 1. Squamous blepharitis with chalazion on the lower lid.



FIG. 2. Chronic ulcerative blepharitis with madarosis.



FIG. 3. Gummatous infiltration of the eyelids with ectropion and ulceration of the lower lid, blepharitis and loss of the lashes. (J. V. Cassady.)



FIG. 4. Chalazion



FIG. 5. Acute hordeolum (with an infected lash at its centre).

[To face p. 486.]

capillarity is impaired. Tears then tend to spill over (*epiphora*), a condition which is accentuated if the punctum becomes everted so that it ceases to lie in accurate contact with the bulbar conjunctiva. The continual wetting of the skin with tears leads to eczema, which is followed by contraction. The condition is made worse by perpetually wiping the eyes, so that eventually *ectropion* develops; this causes still more epiphora and a vicious circle is set up.

The causes of blepharitis are very varied. The patients are usually children debilitated from living in poor hygienic conditions or from diseases such as anæmia, tuberculosis, syphilis or measles. The condition may follow chronic conjunctivitis or be induced by the same causes. Occasionally parasites cause blepharitis—*blepharitis acarica*, due to *demodex folliculorum*, and *phthiriasis palpebrarum*, due to the crab-loose, very rarely to the head-loose. In the latter condition the cilia are covered with black nits, producing an appearance easily recognized when once seen.

Treatment. The local treatment of blepharitis must be energetic in the ulcerative form. The crusts must first be removed and loose, diseased lashes epilated. This is effected most easily by thorough bathing with hydrogen peroxide or warm bicarbonate of soda lotion, 3 per cent. The application softens the deposits, so that they can be picked or rubbed off with a pledget of cotton-wool. When the crusts have been entirely removed the surface is covered with an antibiotic ointment depending on the sensitivity of the organism. When the infection has been eliminated stimulating applications may be applied—an aniline dye, an ointment of yellow oxide of mercury, ammoniated mercury, or ichthyol (5 per cent.), which is gently but well rubbed in for at least five minutes, so as to insinuate it into the hair follicles. These procedures should be repeated three times a day. In most cases, if the treatment is carried out properly, there is a speedy cure, but unfortunately the treatment is seldom carried out satisfactorily. It is useless merely to smear ointment on the surface of the crusts; it must be applied to the inflamed tissues and rubbed well into the lashes. This treatment should be continued for two or three weeks after apparent cure, as organisms lie hidden in the follicles and the inflammation is likely to recur.

In more chronic forms when the active infection is brought under control, stimulatory treatment may be advisable—daily painting of the lid margins with 20 per cent. protargol, 2 per cent. silver nitrate, 0.5 per cent. brilliant green or crystal violet. Attention must be directed to improving the hygiene of the surroundings and the general health; autogenous vaccines may be useful.

Syphilis. A *primary chancre* near the lid-margin is a rarity; it may be caused by a kiss and occasionally by the unsavoury practice of removing a foreign body with the tongue. There is generally a small ulcer covered with scanty greyish secretion, much indurated about the

base, considerable œdema and involvement of the regional lymph nodes. The swelling of the lymph nodes is always suggestive of syphilis or tuberculosis, but in all doubtful cases scrapings should be examined for treponemata. General penicillin treatment or other energetic constitutional measures should at once be undertaken.

Gummata sometimes occur in the lids, and occasionally may cause enormous thickening of the tarsus (*syphilitic tarsitis*) (Plate XXI, Fig. 3). An isolated gumma may be mistaken for a chalazion. In syphilitic tarsitis the lid may be so swollen and hard that it is impossible to evert it. The pre-auricular lymph node is swollen. If the onset is slow there is little pain; sometimes the swelling is rapid and very painful. Gummata usually respond rapidly to appropriate anti-syphilitic treatment with penicillin or even mercury and iodides.

Vaccinia. The margin of the lid is occasionally inoculated from the recently vaccinated arm of a baby, and one lid may infect the opposing margin of the other. Usually the pustule is at the outer canthus, and the pre-auricular lymph node is swollen and painful. The history generally serves to elucidate the case. Sometimes the cornea becomes affected. *Treatment* is outlined on p. 208.

INFLAMMATION OF THE GLANDS OF THE LIDS

Hordeolum or sty is a suppurative inflammation of one of Zeis's glands (Plate XXI, Fig. 5). In the early stages the gland becomes swollen, hard and painful, and usually the whole edge of the lid is œdematous. An abscess forms which generally points near the base of one of the cilia.

The pain is considerable until the pus is evacuated. Styes often occur in crops, or may alternate with boils on the neck, carbuncles, or acne, usually indicating a deficient resistance to staphylococci. It is commonest in young adults, but may occur at all ages, especially in debilitated persons.

Treatment. Hot compresses should be used in the early stages. When the abscess points it may often be evacuated by pulling out the affected cilium; alternatively, it is incised with a small knife, which may be momentarily painful. Thereafter a hot compress is applied. Antibiotic ointments may prevent recurrences.

If crops of styes occur the general health must receive attention, and conditions such as diabetes excluded. A general course of antibiotic treatment may stop recurrences if they have not persisted long, but in obstinate cases a staphylococcal vaccine, preferably autogenous combined with toxoid, may be used.

Hordeolum internum, a suppurative inflammation of a meibomian gland, is less common; it may be due to secondary infection of a chalazion. The inflammatory symptoms are more violent than in an external sty, for the gland is larger and is embedded in dense fibrous tissue. The pus appears as a yellow spot shining through the conjunctiva when the lid is everted. It may burst through the duct or the conjunctiva, rarely through the skin. *Treatment* is the same as for the

external type, except that the incision should be made exactly as for a chalazion (*vide infra*).

Chalazion (*Tarsal "Cyst", Meibomian "Cyst"*) is not a cyst but a chronic inflammatory granuloma of a meibomian gland. Chalazia are often multiple, occurring in crops, a condition commoner in adults than in children. The glandular tissue becomes replaced by granulations containing giant cells, probably as a result of chronic

FIGS. 402-406. Special instruments for lid surgery (two-thirds size).
(Weiss).



FIG. 402. Beer's knife.



FIG. 403. Sharp spoon.



FIG. 404. Chalazion clamp.



FIG. 405. Protective spatula.



FIG. 406. Lid-clamp.

irritation by an organism of low virulence. The gland becomes swollen, increasing in size very gradually and without inflammatory symptoms so that patients usually seek advice on account of the disfigurement (Plate XXI, Fig. 4). The smaller chalazia are difficult to see, but are readily appreciated by passing the finger over the skin. If the lid is everted the conjunctiva is red or purple over the nodule, in later stages often grey, or rarely, if infection has

occurred, yellow (*hordeolum internum*). The grey appearance is due to alteration in the granulation tissue which becomes converted into a jelly-like mass. Complete spontaneous resolution rarely occurs. The contents may be extruded through the conjunctiva, and in these cases a fungating mass of granulation tissue often sprouts through the opening, keeping up conjunctival discharge and irritation. Sometimes the granulation tissue is formed in the duct of the gland, from which it projects as a reddish-grey nodule on the intermarginal strip (*marginal chalazion*).

Treatment. Chalazia should be incised and thoroughly scraped.

The conjunctival sac is well anæsthetized, and the lid with a regional injection of Procaine, the lid everted and at the point of greatest discoloration a vertical incision is made through the palpebral conjunctiva with a sharp scalpel or Beer's knife (Fig. 402). Any semi-fluid contents which may be present escape and the walls of the cavity are thoroughly scraped with a small sharp spoon (Fig. 403). Bleeding soon stops and no dressing is usually necessary. A clamp (Fig. 404) is a useful adjunct in the manipulations.

The patient should be warned that the swelling will remain for some time since the cavity becomes filled with blood. Sometimes, especially if the scraping has not been sufficient, granulation tissue sprouts from the wound. This must be snipped off with scissors and the cavity again scraped out.

Very hard chalazia are occasionally met with, particularly near the canthi; these may be adenomata of the glands and require excision. Malignant changes occur but are rare; in all cases wherein recurrences persist, however, a histological section should be examined.

A marginal chalazion with granulation tissue protruding from the mouth of the gland is best treated by diathermy, with a small needle as the active electrode. A current of 20–30 milliamperes is passed for one second, and the operation repeated if necessary.

ANOMALIES IN THE POSITION OF THE LASHES AND LIDS

Trichiasis (*θρίξ*, *τρίχος*, a hair) is the condition of distortion of the cilia, so that they are directed backwards and rub against the cornea. Any condition causing entropion (*q.v.*) will cause trichiasis, trachoma and spastic entropion being among the most common; other causes are blepharitis, and the scars resulting from injuries, burns, operations, or destructive inflammations such as diphtheria. A few of the lashes may be affected, or the condition may be due to entropion involving the whole margin of the lid. It may also be caused by congenital distichiasis (*q.v.*).

The symptoms are those of a foreign body continually present in the eye—irritation, pain, conjunctival congestion, reflex blepharospasm and lacrimation. Recurrent erosions, superficial opacities and vascularization of the cornea are eventually produced; recurrent corneal ulcers are not infrequently due to this cause.

Treatment. Isolated misdirected cilia may be removed by epilation but this must be repeated every few weeks. Destruction of the hair follicle by diathermy or electrolysis is preferable.

With the former, a fine needle is inserted into the hair follicle and a current of 30 milliamperes is applied for ten seconds. With the latter, the flat positive pole is applied to the temple; the negative, a fine steel needle, is introduced into the hair follicle: a current of two milliamperes is used. The negative pole is determined by placing the terminals in saline, when bubbles of hydrogen are given off by it. The strength of current can be gauged by the rate of evolution of gas. It should be remembered that electrolysis is both painful and tedious; the pain may be avoided by injecting Procaine into the margin of the lid. If the current is of the proper strength, the bubbles produced at the site of puncture cause the formation of a slight foam, and the lash with its bulbous root can be easily lifted out.

If many cilia are displaced, resort must be had to operative procedures as for entropion (*vide infra*).

Entropion (ἐν, in, *τρέπειν*, to turn), rolling in of the lid, occurs in two forms, spastic and cicatricial. The symptoms are those of the trichiasis (*q.v.*) which is induced.

Spastic entropion is due to spasm of the orbicularis. Strong contraction of the circularly arranged fibres tends not only to approximate the lid margins, but also to turn them inwards or outwards, according to the degree of mechanical support afforded by the globe and orbital contents. If the support is insufficient, entropion is produced. This is well seen when the eyeball has been removed, but it also occurs when the globe is deeply set owing to absence of orbital fat, especially if the skin of the lids is also redundant. These conditions are found particularly in old people who are therefore liable to spastic entropion. It is also caused by tight bandaging as after a surgical operation, and is favoured by narrowness of the palpebral aperture (blepharophimosis). Spastic entropion is almost invariably restricted to the lower lid (Fig. 407).

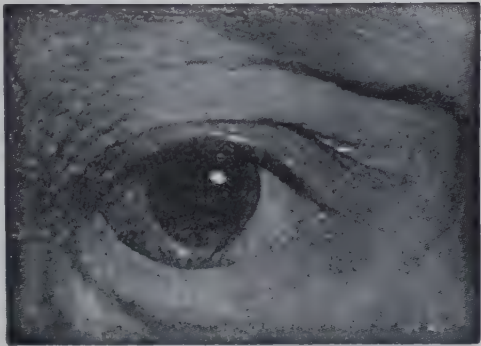


FIG. 407. Spastic entropion. The lid margin is partially curled in and the lashes sweep the cornea.

Cicatricial entropion is caused by cicatricial contraction of the palpebral conjunctiva; in the worst forms, found in trachoma, the tarsal plate is also bent and distorted, sometimes by atrophic, sometimes by hyperplastic changes. It is an exaggeration of the effect produced by the various causes of trichiasis (*q.v.*).

Treatment of Spastic Entropion. If due to bandaging, the condition is often cured by simply leaving off the bandage. In the spastic entropion of old people temporary relief may be obtained by everting the lid, by painting collodion on the skin, by pulling it out with a strip of adhesive plaster or by a subcutaneous stitch. Injection of 1 ml. of 80 per cent. alcohol subcutaneously along the edge of the lid,

with or without canthoplasty, has been advocated. Permanent relief can be obtained only by operation.

The simplest method is the removal of a strip of skin and muscle. Under regional anæsthesia an oval area of skin, with the long axis horizontal and varying in width according to the amount of entropion and of superfluous skin, is marked out just below the lid-margin. The strip of skin is removed and the underlying fibres of the orbicularis, particularly the fibres of Riolan just at the lid margin, dissected out until the tarsus is exposed. Four or five sutures should be inserted.

In cases of spastic entropion with much blepharospasm, *canthoplasty* is sometimes indicated. It consists in widening the palpebral aperture by dividing the outer canthus after the injection of an analgesic. The lids are separated with the fingers in such a manner as to put the canthus on the stretch and the commissure exsanguinated by clamping it firmly for one minute in artery forceps. The entire thickness, including skin and conjunctiva, is then divided horizontally by a single cut with blunt-pointed scissors. If only a temporary effect is required no sutures are inserted; if it is desired permanently to enlarge the palpebral aperture, the conjunctiva is sutured to the skin. Temporary canthoplasty is sometimes indicated in conditions other than spastic entropion, e.g., in simple severe blepharospasm, such as occurs in phlyctenular conjunctivitis, in acute purulent conjunctivitis with much swelling of the lids, in operations for retinal detachment, especially scleral resection, and in removal of an enlarged eyeball or an orbital tumour.

Treatment of Cicatricial Entropion. Many plastic operations have been devised for the relief of cicatricial entropion: only the more simple will be described here. The principles governing the various operations are: (1) altering the direction of the lashes, (2) transplanting the lashes, (3) straightening the distorted tarsus. Subcutaneous injection of Procaine or a general anæsthetic is indicated; the former method does not obviate all pain, especially if the tarsus is cut.

The simplest procedure is some modification of *Burow's operation*. The lid is everted over the end of a metal lid spatula (Fig. 405). A horizontal incision through the conjunctiva and passing completely through the tarsal plate, but not through the skin, is made along the whole length of the lid in the sulcus subtarsalis, about 2-3 mm. above the posterior border of the intermarginal strip (Fig. 408). The temporal end of the strip may then be divided by a vertical incision through the free edge of the lid, including the whole thickness. In this manner the edge of the lid is left attached only by skin, and when cicatrization has occurred the edge is turned slightly outwards, so that the lashes are directed away from the eye. The edge of the lid may be kept everted during the process of healing by means of a spindle-shaped pad of oiled silk kept in position by sutures suitably applied. Relapses are not uncommon, however, and the operation may have to be repeated.

Fig. 409 illustrates an alternative operation. The incision is made as before. The tarsal plate is pared down to a chisel-edge along the whole length and mattress sutures are passed through the plate and lid margin, emerging through the grey line (p. 483); they are tied over rubber tubing, thus bending the lid margin forwards and upwards.

In the *Jaesche-Arlt operation* the zone of hair follicles is transplanted to a slightly higher position. The globe is protected by the spatula inserted between it and the lid, or held by a lid clamp (Fig. 406). The lid is split from the outer canthus to just outside the punctum along the grey line.

The incision extends between the tarsus and the orbicularis for a depth of 3-4 mm., so that the zone containing the hair follicles is loosened (Fig. 410). A crescentic piece of skin is then removed from the lid. The lower incision extends through the skin down to the tarsus at a distance of 3-4 mm. from the edge of the lid and parallel with it for its whole length. The middle part of

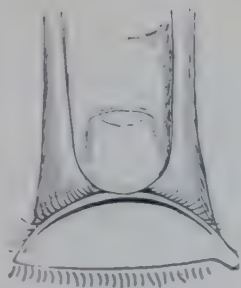


FIG. 408. Incision for Burow's operation for entropion. The upper lid is everted over the spatula.



FIG. 409. Diagram of modified Burow's operation for entropion to show the final position of the lid margin.

the upper incision is 6-8 mm. from the edge of the lid. The crescentic piece of skin thus marked out is removed, without taking any orbicularis. The two skin incisions are then sutured (Fig. 411). In this manner the zone of lashes is transplanted to a higher level. The gaping wound in the intermarginal strip may be filled in with a graft of mucous membrane from the lip; this tends to prevent the follicles from being drawn down again when the wound cicatrizes. Care should be taken not to produce ectropion by removing too much skin.

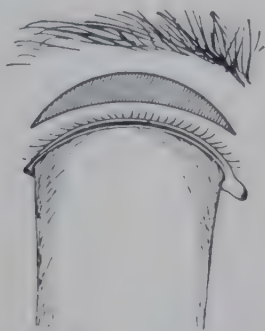


FIG. 410.

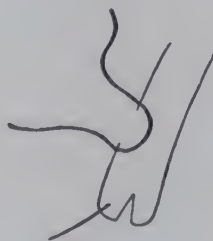


FIG. 411.

FIGS. 410 and 411. Diagram of Jaesche-Arlt operation for entropion.

Ectropion, rolling out of the lid, occurs in several forms, the chief being spastic, cicatricial, senile, and paralytic. The symptoms are due to the epiphora induced and to the chronic conjunctivitis caused by exposure. In long-standing cases the exposed conjunctiva becomes dry and thickened, red and very unsightly and in severe cases the cornea may suffer from imperfect closure of the lids.

Spastic ectropion results from blepharospasm when the lids are well supported by the globe and when they are short, firm, and without redundant skin. It is therefore seen in children and young

patients. Mechanical ectropion is caused by thickening of the conjunctiva, as occurs after purulent conjunctivitis and trachoma. Upper and lower lids may be affected simultaneously.

Cicatricial ectropion results from destruction of the skin by chronic conjunctivitis or blepharitis, injury, burns, ulcers, gangrene or operations (Fig. 412); tuberculous caries of the orbital bones is a rare cause in children.



FIG. 412. Cicatricial ectropion from a burn.

Senile ectropion is found only in the lower lid, and is due to relaxation of the tissues and laxity of the fibres of the orbicularis muscle. The condition is increased by the conjunctivitis and epiphora which are set up (see Fig. 426).

Paralytic ectropion results from the laxity of the lids induced by paralysis of the orbicularis. Only the lower lid is affected, the upper being kept in contact with the globe by its own weight.

Treatment. Non-operative treatment is of little use except in spastic ectropion. Here a well-fitting bandage, unless contra-indicated by other factors, will often cure the displacement. In paralytic ectropion, the condition is cured only by restoration of the innervation. The slighter degrees of senile ectropion are also amenable to non-operative treatment, although it may be advisable to slit the canaliculus in order to cure epiphora (p. 509). The patient should be instructed not to pull the lid down when wiping the eye. A large variety of operations has been devised for ectropion: only the simpler procedures will be described.

In paralytic ectropion *lateral tarsorrhaphy* may be indicated. In this operation the palpebral aperture is shortened by uniting the lids at the outer canthus. The edges of the upper and lower lids are freshened for the requisite distance, the lashes being excised. The lids are then sutured together as in central tarsorrhaphy (p. 414).

In many cases of ectropion, especially senile, the lower lid is stretched and elongated. The ectropion may then be cured by *shortening the lid* as in Dimmer's modification of Kuhnt's operation (Figs. 413, 414). A triangular piece of conjunctiva and tarsus is excised, the apex of the triangle being towards the fornix and the lid split along the grey line from the triangle to the outer canthus. A triangular area of skin is removed at the outer canthus and the skin is slid outwards so that the gap in the tarsal plate is closed, the

requisite length of the margin of the lid at the outer canthus being denuded of cilia. Care should be taken to carry the upper skin incision up and out, so that the lid will be drawn slightly upwards. It is quite as effective to remove the triangle of tarsus at the outer canthus, and this avoids the necessity of splitting the lid.

In most of these operations restoration of the normal position is facilitated by dissecting off the strip of thickened conjunctiva at the margin of the lid.

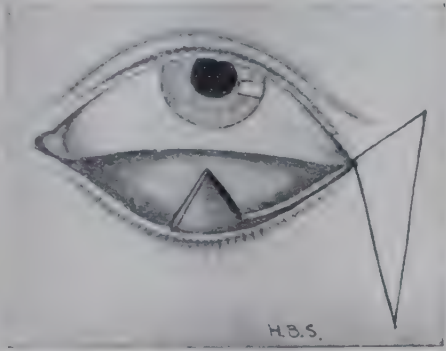


FIG. 413.

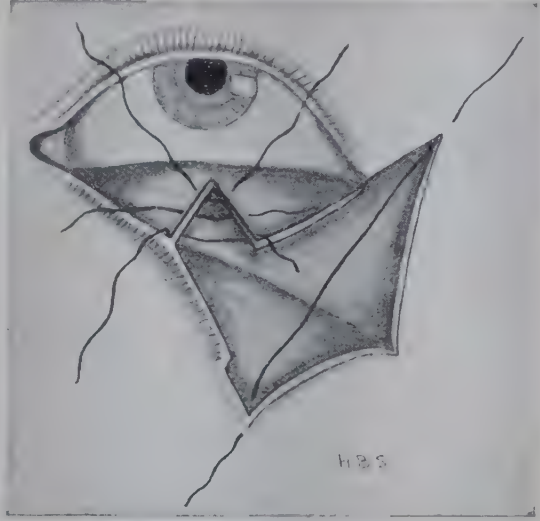


FIG. 414.

FIGS. 413 and 414. Dimmer's modification of Kuhnt's operation for ectropion.

In the slighter cases of cicatricial ectropion the *V—Y operation of Wharton Jones* is indicated (Fig. 415). A V-shaped incision, with the apex away from the lid margin, is made through the skin, the limbs of the V enclosing the cicatrix. The skin is freed from the underlying tissues and well undermined and the margins sutured in such a manner that a Y-shaped cicatrix results.

More extensive cicatricial displacement requires some form of *blepharoplasty*, employing whole or split-skin grafts or flaps of skin taken from the upper lid, behind the ear or the inner upper arm, or strips of fascia lata. Each case must be treated on its own merits and will often exercise the ingenuity of the surgeon.

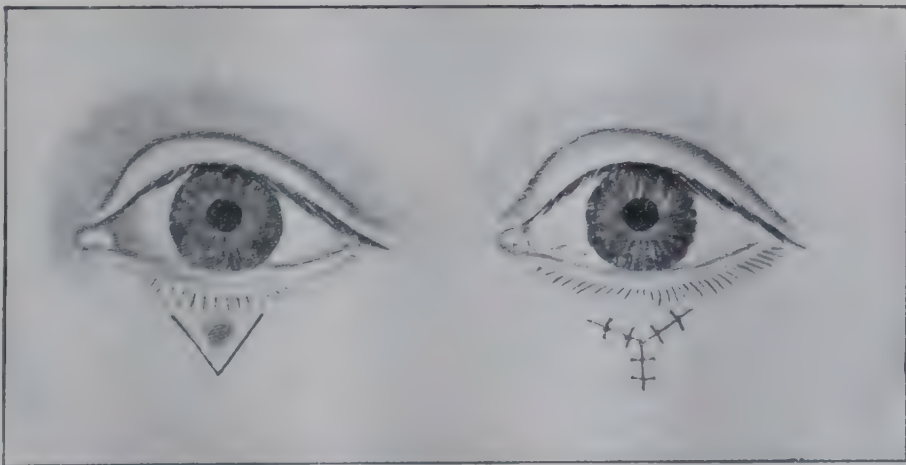


FIG. 415 Diagram of V—Y operation for ectropion.

Symblepharon (σύν, with, together, βλέφαρον, eyelid) is the condition of adhesion of the lid to the globe (Fig. 416). Any cause which produces raw surfaces upon two opposed spots of the palpebral and bulbar conjunctiva will lead to adhesion if the areas are allowed to remain in contact during the process of healing—burns from heat or caustics, ulcers, diphtheria, operations, etc. Bands of fibrous tissue are thus formed, stretching between the lid and the globe,

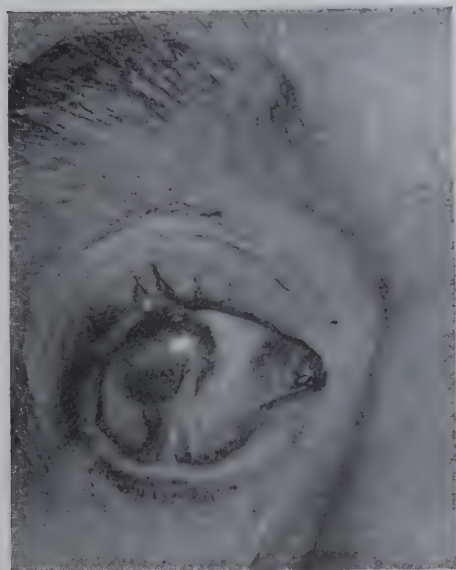


FIG. 416. Symblepharon.

involving the cornea if this has also been injured. The bands may be narrow, but are more frequently broad, and may extend into the fornix so that the lid is completely adherent to the eyeball over a considerable area (*symblepharon posterior*). Bands limited to the anterior parts and not involving the fornix are called *symblepharon anterior*. *Total symblepharon*, in which the lids are completely adherent to the globe, is rare.

Pronounced adhesions cause impairment of mobility of the eye resulting in diplopia. The adhesion may be so intimate that it is impossible to close the lids efficiently, lagophthalmos with its baneful consequences resulting (*vide infra*). There is often much disfigurement.

Treatment. The prevention of symblepharon is of the utmost importance (p. 374). When it is already established operation is necessary and this may be difficult especially when the bands are broad or if there is *symblepharon posterior*. There may be no guide to the limitations of sclera and tarsus, and great care has to be exercised lest the globe be punctured. The prevention of the re-formation of adhesions is much more difficult, and is successful only if the raw surfaces are covered with conjunctival or buccal mucous membrane grafts.

Ankyloblepharon (ἀγκύλη, a thong, βλέφαρον, eyelid) is adhesion of the margins of the two lids. It may be either a congenital condition or due to burns, etc. It may be partial or complete, and is often combined with symblepharon. The treatment depends upon the amount of symblepharon. If it is very extensive operation may be contra-indicated. In other cases the lids are separated and kept apart during the healing process. If the adhesion extends to the angle of the lids, the latter must be covered with an epithelial graft, otherwise the condition will recur.

Blepharophimosis (βλέφαρον, eyelid, φμός, a muzzle) is the condition in which the palpebral fissure appears to be contracted at the

outer canthus. The outer angle is often normal, but is obscured by a vertical fold of skin formed by eczematous contraction of the skin following prolonged epiphora and blepharospasm (*epicanthus lateralis*). Mere narrowing of the palpebral aperture is often called blepharophimosis, and may be a congenital condition: it is really a form of ankyloblepharon. The condition may require no treatment, disappearing spontaneously after the inflammation has subsided. In other cases canthoplasty is indicated.

Lagophthalmos (λαγῶς, a hare) is the condition of incomplete closure of the palpebral aperture when an attempt is made to shut the eyes. It may be due to contraction of the lids from cicatrization or a congenital deformity, ectropion, paralysis of the orbicularis, proptosis due to exophthalmic goitre, orbital tumour, etc., or to laxity of the tissues and absence of reflex blinking in people who are extremely ill or moribund. Owing to exposure the cornea becomes epidermoid (xerosis corneæ) or keratitis sets in. The treatment is that of keratitis e lagophthalmo (*q.v.*).

Ptoxis (πτῶσις, falling) is the term given to drooping of the upper lid, usually due to paralysis or defective development of the levator palpebræ superioris. It may also occur as a purely mechanical ptosis due to deformity and increased weight of the lid brought about by trachoma or tumours, or from lack of support in phthisis bulbi or anophthalmos.

The condition may be unilateral or bilateral, partial or complete. In the higher degrees the lid hangs down, covering the pupil more or less completely and interfering with vision. An attempt is made to counteract the effect by overaction of the frontalis and by throwing back the head, the eyes being rotated downwards at the same time. A

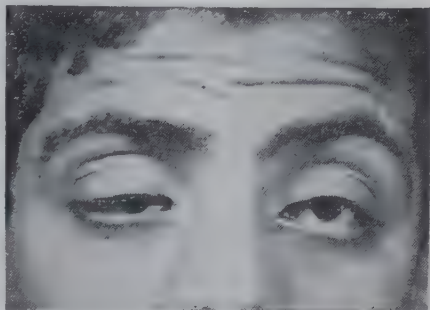


FIG. 417. Congenital ptosis, more marked on the right side. Note the wrinkling of the forehead in the attempt to open the eyes (Kamel).

very characteristic attitude is thus adopted; forced contraction of the frontalis causes the eyebrows to be raised and throws the skin of the forehead into wrinkles (Fig. 417). Partial ptosis may be masked by this means, but becomes manifest if the patient is asked to look up while the eyebrows are fixed by firm pressure with the fingers against the frontal bone.

Ptoxis may be congenital or acquired. The congenital form is usually, but not invariably, bilateral, and is due in most cases to defective development of the muscles. Some cases have been proved to be caused by maldevelopment of the third nucleus. The condition is not infrequently hereditary. There is often an associated defect in the upward movement of the eyes, due sometimes to absence of the posterior insertion of the levator into the

fornix, sometimes to coincident maldevelopment or defective innervation of the superior rectus. Defective upward movement of the eyes is the commonest congenital defect of the bilaterally associated extrinsic muscles. A *periodic ptosis* may be present in association with abnormal synkineses (p. 465).

Acquired ptosis is usually unilateral. It may be part of the symptom-complex of paresis or paralysis of the whole of the third nerve, or may be due to paresis or paralysis of the branch supplying the levator. Isolated ptosis without other signs of oculomotor paralysis may result from disease of the supranuclear pathways (p. 543). It may also be due to direct injury of the muscle or its nerve supply, as by wounds or fractures. Bilateral ptosis may occur in the acquired form, notably as part of the syndrome of myasthenia gravis or occasionally in myotonic dystrophy.



FIG. 418.

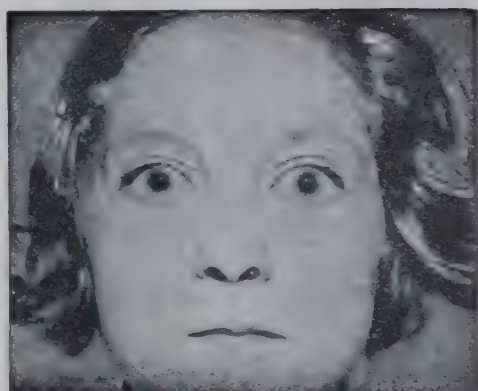


FIG. 419.

FIGS. 418-9. Myasthenia gravis. Fig. 418, the usual condition ; Fig. 419, after the injection of 1 mg. of Prostigmin (F. B. Walsh).

Myasthenia gravis is a disease characterized by generalized muscular weakness and rapidly developing fatigue of the muscles due probably to interference with the activity of the cholinesterase necessary to implement muscular activity at the myoneural junctions. The symptoms fluctuate and after a short rest recovery follows rapidly in the early stages. Ptosis and failure of convergence are early and prominent features. The ptosis is nearly always bilateral and is increased by prolonged fixation or attempts to look upwards ; effective compensation by overaction of the frontales is impossible. Ophthalmoplegia externa, partial or complete, occurs in 50 per cent. of the cases, but the intrinsic muscles are not affected. Nystagmoid jerks are not uncommon. Remarkable temporary improvement in the action of the muscles is obtained by injections of Prostigmin (Figs. 418 and 419).

Treatment. In cases of paralysis of the third nerve treatment must be directed to removal of the cause. The fact that this nerve is frequently affected in syphilis must be borne in mind ; these

cases respond to treatment better than others. In cases of incurable paralysis and in congenital and mechanical ptosis the deformity can be relieved only by operation. In complete paralysis of the third nerve, operation is usually contra-indicated on account of the abduction of the eye since if the lid is raised in these cases the diplopia becomes manifest.

Operations for ptosis ameliorate the condition but seldom give perfect results. Three techniques may be applied : (1) if the levator is not completely paralysed this muscle may be shortened ; (2) if the levator is paralysed but the superior rectus is active, the latter muscle may be pressed into service to lift the lid ; (3) if both

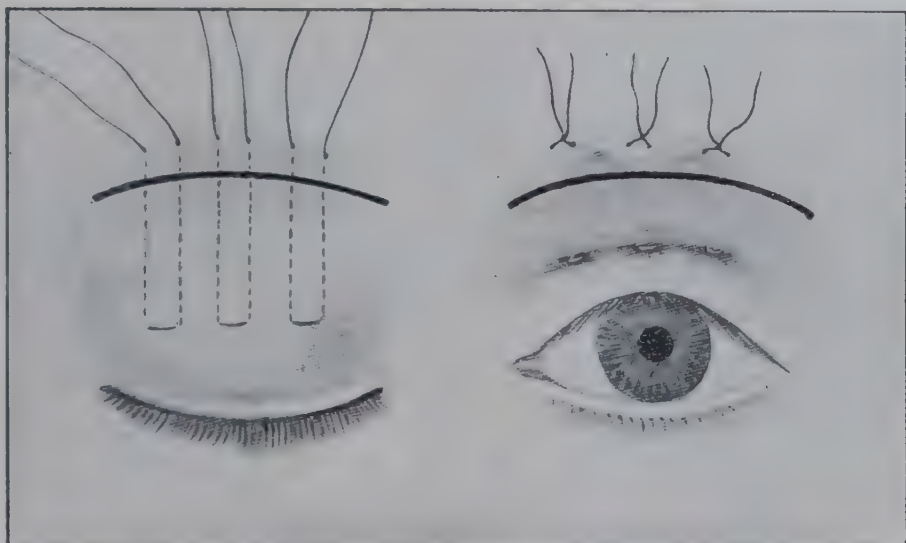


FIG. 420. Diagram of Hess's operation for ptosis.

levator and superior rectus are paralysed, the action of the frontalis muscle may be utilized in raising the lid.

(1) In the *Blaskowicz operation*, the levator muscle is shortened by an approach from the conjunctival surface. The lid is everted, an incision is made down to the upper border of the tarsal plate along its whole width and the conjunctiva reflected above and below to expose the tendon of the muscle and the plate. The tendon is widely freed, controlled by temporary sutures passed through it close to its attachment to the tarsal plate. It is then cut from its attachment and a strip of tarsal plate (4 mm.) removed with scissors. Three double-armed sutures are threaded through the lower edge of the conjunctival incision, passed through the tendon 7 to 10 mm. above its cut border, and the redundant tissue excised. While the muscle is put on the stretch another row of double-armed sutures is passed through it 3 mm. above the line of the previous sutures, brought out through the skin, and tied at a point mid-way between the lower and upper limits of the lid so as to maintain the natural fold in the skin. The previous sutures are then passed through the skin at the new level of the tarsal plate (2 to 3 mm. above the lash margin) and threaded through glass beads.

In *Eversbusch's technique* the same operation is performed from the skin surface. Technically this is more difficult.

(2) In *Motais's operation* the central third of the tendon of the superior rectus is transplanted to the upper border of the tarsal plate through a subconjunctival approach. This operation and its many modifications, however, tend to leave a varying degree of vertical muscular imbalance and are contra-indicated in unilateral cases.

(3) *Hess's operation* is the simplest when both muscles are paralysed (Fig. 420). An incision is made below the eyebrow for 2.5 cm. The skin of the lid is then undermined through this incision so that it is completely separated from the orbicularis and tarsus over the whole area. Three silk sutures are then inserted as shown in the figure. A more effective procedure is to raise the lid in the same manner by narrow strips of fascia lata similarly inserted.

If after these operations it is found that when the eyeball is raised, the skin of the upper lid falls in an unsightly fold over the lashes, a horizontal strip of skin of suitable width is removed from the upper lid, the position of the lower skin incision corresponding roughly to that of the upper edge of the tarsal plate. The sutures which join the edges of the skin incision are carried through the deep tissues in such a way as to stretch the skin over the tarsus and to produce a fold in the skin of the eyelid in the normal position.

TUMOURS OF THE LIDS AND ALLIED CONDITIONS

Benign growths include xanthelasma, molluscum, warts, nævus, angioma, and other tumours common to the skin and cutaneous glands.

Small clear *cysts* frequently occur among the lashes in old people, due to the retention of secretion of Moll's glands. They disappear if the anterior wall is snipped off.

Xanthelasma (ξανθός, yellow, ἐλασμα, a plate) or xanthoma is a slightly raised yellow plaque, most commonly found in the upper and lower lids near the inner canthus, and often symmetrical in the two lids and on both sides. They are most common in elderly women, and are sometimes associated with diabetes and excessive formation of cholesterol. They grow slowly, and only require treatment on account of the disfigurement produced. They may be excised or destroyed by trichloroacetic acid or carbon dioxide snow.

Molluscum contagiosum is a small white umbilicated swelling, generally multiple, due to a virus infection, from which a substance resembling sebum can be expressed. It produces a severe conjunctivitis and occasionally a keratitis which are intractable to treatment unless the primary nodules on the lid margin are dealt with. Each should be incised and expressed and the interior touched with tincture of iodine or pure carbolic acid; systemic treatment by sulphadiazine has given good results.

Nævus or mole, usually pigmented, may occur on the lids, generally affecting the margin and involving both skin and con-

junctiva. Two may be symmetrically situated on the lids of the same eye, indicating their origin at a time when the lids were still united. The microscopical appearance is characteristic, consisting of nævus cells, often arranged in an alveolar manner. They may grow at puberty but very rarely take on malignant proliferation. They may be removed by excision but this ought to be complete and extensive.

Hæmangioma, often also called nævus, occurs in two forms—telangiectases and cavernous hæmangiomata (Fig. 421). The former are bright red or port-wine coloured spots composed of dilated capillaries. The latter consist of dilated and anastomosing vascular spaces lying in the subcutaneous tissue having all the characteristics of erectile tissue; they are not infrequently strictly localized as if partially encapsulated. They appear bluish when seen through the skin and form swellings which increase in size on venous congestion as on crying or lowering the head. Cavernous hæmangiomata are rarely seen in adults, partly owing to the fact that they are generally treated in early life, but possibly due to spontaneous atrophy of the growth and thickening of the skin.



FIG. 421. Large hæmangioma of the lids and cheek (W. H. Brown).

Hæmangioma often follows the distribution of the first and second divisions of the fifth nerve. In the *Sturge-Weber syndrome* it is associated with hæmangioma of the choroid and glaucoma, and also with hæmangioma of the leptomeninges, causing homonymous hemianopia or epilepsy. The intracranial lesion may be diagnosed radiographically since there are often calcareous deposits in the underlying cortex.

Telangiectases and small hæmangiomata usually disappear and may well be left alone; if they increase in size, however, treatment is indicated, if the tumour is small by excision, electrolysis or carbon dioxide snow; if large they are best treated by electrolysis or injections with sclerosing solutions (5 per cent. sodium morrhuate; 10 per cent. double hydrochloride of quinine and urea; or quinine hydrochloride 4, urethane 2, water 30 parts; or simple boiling water). Any residual fibrous mass can readily be excised without excessive hæmorrhage. An alternative treatment is the application of electrolysis in several sittings until the tumour is consolidated with fibrous tissue followed by excision of the mass. Radium therapy is rarely necessary.

Lymphangioma occurs rarely in the lids.

Neurofibromatosis (*Elephantiasis neuromatodes*, *plexiform neuroma*, *von Recklinghausen's disease*) may affect the lids and orbit. In typical cases the temporal region is also affected. The swollen lid and temporal region form a characteristic picture. The hypertrophied nerves can be felt through the skin as hard cords or knobs. The nerve fibres are little changed, the hyperplasia affecting the endo- and peri-neurium. In several cases the ciliary nerves have been found to be affected, both in the orbit, associated with a true glioma of the optic nerve, and inside the globe, which in many cases is buphthalmic. Operative measures are seldom satisfactory. The choroid and ciliary body may be much thickened by layers of dense fibrous-like tissue probably derived from the cells of the sheaths of Schwann. Laminated ovoid bodies resembling Pacchionian corpuscles also occur.

Symmetrical soft swellings above the inner canthus are sometimes seen in elderly people. They are due to prolapse of the orbital fat through an aperture in the orbital septum.

Malignant Tumours include carcinomata, sarcomata and malignant melanomata, the first being much the more common. *Epitheliomata* (squamous-celled carcinomata) show a preference for sites where the character of the epithelium changes ; they therefore commence generally at the edges of the lids. The patients are elderly ; the pre-auricular lymph nodes may be enlarged, or if the growth is near the inner canthus, the submaxillary nodes. Any of the glands of the lid may in rare instances undergo carcinomatous proliferation.

The commonest malignant epithelial growth is the basal-celled

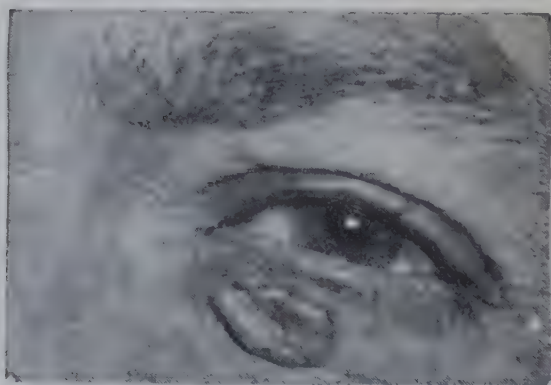


FIG. 422. Rodent ulcer (Smithers).

carcinoma (rodent ulcer), which shows a predilection for the inner canthus (Fig. 422). It commences as a small pimple which ulcerates and if the scab is removed it is found that the edges are raised and indurated. The ulcer spreads very slowly, the epithelial growth extending under the skin in all directions and penetrating deeply. The surrounding structures are gradually destroyed : lids, orbit, and bones are invaded. The growth is only locally malignant and

probably originates in the accessory epithelial structures of the skin—hair follicles and glands—but the lymphatic nodes are not affected. Rodent ulcer rarely occurs before forty years of age, and the rate of growth is usually measured in years.

Sarcoma is rare ; it may be round- or spindle-celled. *Reticular tumours*, round-celled growths variously described as lymphoma, lymphosarcoma, pseudo-leucæmic tumours, etc., sometimes affect both orbits and all four lids causing symmetrical proptosis ; occasionally the patients show blood changes as in leucæmia. The growth is slow but continuous and the eyes may be endangered by lagophthalmos. Malignant melanomata are rare, as also is a more generalized pigmentation developing in adult life which may become cancerous (*melanosis*) (p. 184).

Treatment. Epitheliomata and sarcomata must be thoroughly extirpated by surgery even if it involves excision of the globe or exenteration of the orbit. Rodent ulcer is sensitive to radiation ; if small, however, it is probably best excised ; if so large as not to be amenable to operative treatment without sacrificing a good eye, it may be satisfactorily treated with radium or X-rays provided there is no involvement of the bones. Occasionally, however, the results of radiational treatment may be misleading, for the skin surface may show a firm scar while the growth continues to spread beneath the surface ; in every case it is wise to keep a careful watch for any recurrence. In the later stages extensive plastic operations may have to be performed to protect the eyeball and when this becomes impossible the eye must be excised and the morbid tissues freely removed. Most reticular tumours are radio-sensitive as is melanosis in its pre-cancerous phase, although by no means always in its cancerous phase.

INJURIES OF THE LIDS

Injuries of the lids of various kinds—contusions, wounds, burns, etc.—are very common. They must be treated upon general principles but special attention must be directed to associated injuries of the bones of the orbit (p. 531) and the eyeball.

Contusions are often more alarming in appearance than in reality (the "black eye"). There is great swelling and ecchymosis both in the lids and conjunctiva. In all cases a guarded prognosis should be given, for it may be impossible to determine the full extent of the injury to the orbit or the eye.

Wounds in the direction of the fibres of the orbicularis gape little and heal without conspicuous scarring ; hence surgical wounds should be made in this direction as often as possible. Vertical wounds gape causing disfiguring cicatrices, and often lead to ectropion or other distortion, especially if there is adhesion to the subjacent bone. The worst wounds are those which sever the lid

vertically in its whole thickness. If they do not unite by first intention, a notch (traumatic coloboma) is left in the lid margin, and disfigurement, lagophthalmos and epiphora result.

Treatment. Simple contusions with ecchymosis require only conservative treatment with cold compresses. Wounds must be thoroughly cleansed and brought together by sutures. On account of the rich blood-supply it is not necessary to make a wide excision of the edges : only obviously contused and devitalized tissue should be excised. As a prophylactic against infection the wound should be dusted with sulphonamide and penicillin powder. Lacerated wounds are likely to leave ugly scars and deformities : these must be treated by plastic operation. If suppuration occurs the abscess must be opened and treated on general surgical principles. Vertical wounds severing the canaliculus require special care (Chapter 32).

Burns. It is important to diagnose the degree of a burn. First degree burns require cleansing and the application of sterile saline and penicillin packs every three hours during the day. Second degree burns should be cleansed, any vesicles opened and dead epithelium removed ; subsequent treatment is similar. Third-degree burns should be cleaned and immediately covered by whole- or split-skin grafts, a temporary tarsorrhaphy being performed and allowed to remain until all risk of cicatricial ectropion has passed. This is the great danger from burns of the lids that they easily lead to a severe exposure keratitis with permanent impairment or even loss of vision (Fig. 412). For this reason coagulants (tannic acid, etc.) should never be applied to burns of the lids. The best prophylactic is grafting before scar tissue has formed ; if the initial graft does not take well it can be repeated at leisure. If scarring has developed cicatricial deformities resulting from burns are corrected by plastic operation.

CONGENITAL ABNORMALITIES OF THE LIDS

Symblepharon, ankyloblepharon, ectropion, entropion and trichiasis occur occasionally as congenital malformations. Ptosis is a fairly common congenital defect.

Distichiasis (δῖς, double, στίχος, a row) is a rare condition in which there is an extra posterior row of cilia, occasionally in all four lids. The posterior row occupies the position of the meibomian glands which are reduced to ordinary sebaceous glands performing the normal function of lubricating the hairs ; these lashes may irritate the cornea.

Coloboma of the lid is a notch in the edge of the lid (Fig. 423). The gap is usually situated to the inner side of the middle line, generally affecting the upper lid but two or more defects may occur in the same lid. Sometimes a bridge of skin links the coloboma to the globe, or there is a dermoid astride the limbus at the site of the coloboma.

There are often other congenital defects of the eye or other parts of the body such as coloboma of the iris or accessory auricles. Some cases are due to incomplete closure of the embryonic facial cleft, others probably to the pressure of amniotic bands. Occasionally there is a notch at the outer part of the lower lid, associated with maldevelopment of the first visceral arch (*mandibulo-facial dysostosis*).



FIG. 423. Coloboma of the upper lid (Reese).

Cryptophthalmos (*κρυπτός*, hidden) is a very rare condition in which skin passes continuously from the brow over the eye to the cheek, associated with abnormalities of the eye and often of the orbit.

Microblepharon is the condition in which the lids are abnormally small; they may be absent or virtually so—*ablepharon*. These conditions usually occur only in cases of microphthalmos or congenitally small eyes. Microphthalmos may be associated with a congenital *orbito-palpebral cyst*, causing a swelling of the lower lid. The cyst is connected with the eyeball, contains retinal tissue in its lining, and is due to defective closure of the embryonic fissure—an extreme case of ectatic coloboma of the choroid (*q.v.*). The eyeball may be apparently absent (*congenital anophthalmos*), but there are always microscopic vestiges of ocular tissues.

Epicanthus is a semilunar fold of skin, situated above and sometimes covering the inner canthus (Fig. 424). It is usually bilateral and gives the appearance that the eyes are far apart and have a convergent squint and the bridge of the nose is flat. It may disappear as the nose develops. It is normal in Mongolian races. The deformity can be remedied by plastic surgery.



FIG. 424. Epicanthus.

CHAPTER 32

DISEASES OF THE LACRIMAL APPARATUS

Anatomy and Physiology. The lacrimal apparatus consists of the lacrimal glands and the lacrimal passages.

The *lacrimal glands* of each eye consist of the superior or orbital gland, the inferior or palpebral gland, and the accessory lacrimal glands or Krause's glands. All are serous acinous glands, scarcely distinguishable microscopically from serous salivary glands with which they are morphologically identical. The orbital gland, about the size of a small almond, is situated in the lacrimal fossa at the outer part of the orbital plate of the frontal bone; ten or twelve *lacrimal ducts* pass from it to open upon the surface of the conjunctiva at the outer part of the upper fornix. The palpebral gland consists of only one or two lobules situated upon the course of the ducts of the superior portion. It can be seen when the eye looks down and in after the upper lid has been everted. The accessory or Krause's glands are microscopic groups of acini, lying below the surface of the conjunctiva between the fornix and the edge of the tarsus (Fig. 399). There are about forty-two in the upper, six to eight in the lower fornix. The ducts of numerous acini unite to form a larger duct which opens into the fornix.

The *lacrimal passages* consist of the lacrimal puncta, the canaliculi, the lacrimal sac, and the nasal duct (Fig. 425). The *lacrimal puncta* lie near the posterior border of the free margin of the lid about 6 mm. from the inner canthus. Each lid has one punctum and one canaliculus. The punctum is situated upon a slight elevation, larger in elderly people, the *lacrimal papilla* (Fig. 400). As already mentioned, this is visible in normal circumstances only when the lid is slightly everted (p. 98). The *canaliculus* passes from the punctum to the lacrimal sac; it is first directed vertically for about 1 to 2 mm., then horizontally for 6 to 7 mm. The canaliculi usually open separately through the outer wall of the lacrimal sac. The *lacrimal sac* lies in the lacrimal fossa formed by the lacrimal bone; when distended it is about 15 mm. long vertically, and 5 to 6 mm. wide. The upper portion or *fundus* extends slightly above the level of the inner tarsal ligament and the sac itself is surrounded by fibres of the orbicularis muscle. The lower end narrows as it opens into the *nasal duct*, a tube varying much in size (12 to 24 mm. long, 3 to 6 mm. in diameter) passing downwards and slightly outwards and backwards, bounded by the superior maxilla and inferior turbinate, to open at the anterior part of the outer wall of the inferior meatus of the nose. The line of the duct is given by a

point just outside the inner canthus and the groove between the ala of the nose and the cheek. The upper end of the nasal duct is the narrowest part. The canaliculi are lined by stratified epithelium, the lacrimal sac and nasal duct by columnar epithelium lying upon a corium which contains a venous plexus. The mucous lining forms an imperfect valve at the orifice into the nose.

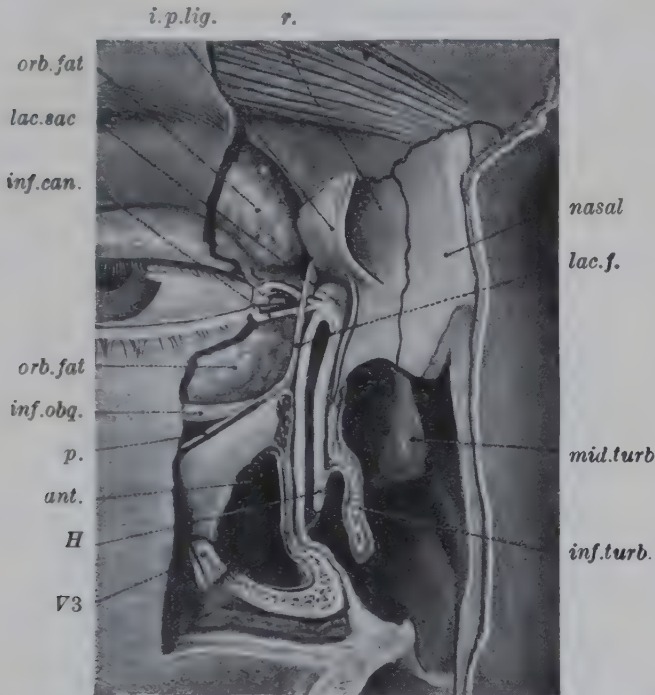


FIG. 425. Canaliculi, lacrimal sac and nasal duct. V3, infra-orbital nerve; H, valve of Hasner; ant., antrum; inf.obq., inferior oblique; orb.fat., orbital fat; inf.can., inferior canaliculus; lac.sac, lacrimal sac; i.p.lig., internal palpebral ligament (turned up); fr., frontal process of superior maxilla; nasal, nasal bone; lac.f., lacrimal fascia; mid.turb., middle turbinate bone; inf.turb., inferior turbinate bone; p., periorbita. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

The *lacrimal secretion* is a slightly alkaline fluid containing sodium chloride as its chief constituent. The ordinary amount secreted is just sufficient to moisten the eyeball, and is lost by evaporation. Only under reflex irritation, psychical or peripheral, is an excess secreted, and this is sucked into the lacrimal sac and forced through the nasal duct into the nose during the act of blinking, when the fibres of the orbicularis contract around the sac. It must be remembered that xerosis or dryness of the conjunctiva does not result from extirpation of the superior and inferior lacrimal glands, the moistening of the conjunctiva by Krause's glands and its own mucous cells being sufficient to prevent it. On the other hand, epiphora does not usually result from extirpation of the lacrimal

sac, except in the presence of psychical or peripheral stimuli to increased secretion. The tears have some slight bacteriostatic properties owing to the presence of an enzyme, lysozyme.

DISEASES OF THE LACRIMAL GLAND

Diseases of the lacrimal gland are rare. *Dacryo-adenitis* occurs occasionally in general infections (mumps, influenza, etc.), sometimes leading to suppuration. Tuberculous infection may occur. A permanent *fistula* may result from the bursting of an abscess in the gland. Spontaneous and traumatic *dislocations* of the gland have been described, a swelling being formed under the outer part of the upper lid.

Dacryops is a cystic swelling in the upper fornix due to retention of secretion owing to blockage of one of the lacrimal ducts. It can only be distinguished from retention cysts of Krause's glands by its position.

Mikulicz's syndrome is characterized by symmetrical enlargement of the lacrimal and salivary glands. The ætiology varies but the swelling is usually of a lymphomatous nature. Both parotid and lacrimal glands are enlarged in uveoparotid inflammation (p. 252).

Tumours of the lacrimal gland show a very marked resemblance to those of the parotid. Much the commonest is a *pleomorphic adenocarcinoma*, frequently characterized histologically by myxomatous material (the so-called "mixed tumour"). It is slow-growing but infiltrative and tends to recur even after apparently successful surgical removal particularly when extensions have invaded the orbital bones.

All conditions which cause swelling of the gland may lead to impairment of movement of the eye. The globe is pushed downwards and inwards; movement outwards, and especially outwards and upwards, is limited. There may be some proptosis.

The rare diseases mentioned above are treated on general principles.

Kerato-conjunctivitis sicca (*Sjögren's syndrome*), a general systemic disturbance of unknown origin usually occurring in women after the menopause and often associated with polyarthritis, is characterized by deficiency of the lacrimal secretion leading to dryness of the eyes. This usually gives rise to chronic irritative symptoms and may be associated with epithelial erosions or filaments on the corneal surface; drying of the conjunctiva may be demonstrated clinically by staining with rose bengal. Pathologically, the lacrimal gland is found to be fibrotic; similar changes in the salivary glands may lead to a dryness of the mouth, while desiccation may occur in other mucous membranes. No effective treatment is known, but sealing of the puncta by diathermy in order to conserve what secretion remains and the frequent use of bland lotions or artificial tears (p. 562) may help to bring comfort.

DISEASES OF THE LACRIMAL PASSAGES

Eversion of the lower punctum occurs from laxity of the lids in old age, from chronic conjunctivitis or blepharitis or any cause

leading to ectropion (*q.v.*) (Fig. 426). If on clinical examination the punctum is visible when the lower lid apposes the globe it may be considered to be everted. This causes epiphora, which in turn aggravates the condition.

Treatment. In slight cases, especially in old people, the eversion may be sufficiently counteracted by burning a deep gutter in the fornix just behind and below the position of the punctum with diathermy. As the cicatricial tissue contracts the punctum is pulled inwards towards the eye.

If this fails the canaliculus should be slit, the object being to convert it into a groove the inner part of which comes into apposition with the globe; it is therefore most important that the canaliculus should be slit through its *posterior* wall. This should only be done if it is necessary and it should never be slit *more* than is necessary, rarely more than 2 mm.



FIG. 426. Eversion of the punctum in a case of senile ectropion.

The simplest method is the so-called "three-snip" operation. The conjunctival sac is anæsthetized and Procaine injected into the tissues around the canaliculus. The punctum is dilated with Nettleship's dilator (Fig. 427) which is introduced vertically and then pushed inwards along the canaliculus. A canaliculus knife (Fig. 428) is then taken and the probe-point is passed into the punctum in the same manner, first downwards, then inwards, the back of the knife being directed forwards and slightly downwards. In this manner, as the knife is pushed inward, the posterior wall of the canaliculus is incised. While this manœuvre is being performed the lid is kept stretched outwards, so that the wall of the duct is kept taut against the edge of the knife. The triangular flap of the posterior wall formed between the vertical and horizontal parts of the canaliculus is then snipped off with scissors. A probe should be passed on the day following the operation, and occasionally on succeeding days, so as to prevent closure of the incision.

In cases of marked eversion of the lower punctum a radical operation for ectropion may be necessary.

Occlusion of the puncta may be congenital, which is extremely rare, or cicatricial. In either case epiphora is caused which is very difficult to treat. Before treatment is commenced the

patency of the lacrimal passages should be ensured by syringing through the other (upper) punctum.

An endeavour should be made to slit up the occluded punctum—not the whole canaliculus. On inspection no trace of the punctum may be visible, but it is rare if some evidence of its presence cannot

FIGS. 427-32. Special lacrimal instruments (two-thirds size except Fig. 428—full-size) (Weiss).



FIG. 427. Nettleship's canaliculus dilator.



FIG. 428. Tweedy's canaliculus knife.



FIG. 429. Couper's probes.



FIG. 430. Lacrimal syringe.

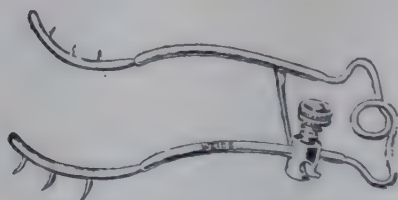


FIG. 431. Briggs's lacrimal retractor.



FIG. 432. Lacrimal dissector.

be seen on minute examination of the normal site with a loupe. The point of the dilator is inserted at this site, and may succeed in opening up the punctum sufficiently to admit the probe-point of the canaliculus knife. There is usually no difficulty in knowing when the knife is in the duct, as it passes on in the proper direction

quite easily. If this method fails to permit an entrance the canaliculus may be cut across vertically. When bleeding has stopped, the outer cut end is examined with a loupe, and the probe point of the knife is inserted into it. Apposition of the wound must be accurate and a nylon thread is left in the canaliculus to ensure continuity in healing.

Occlusion of the canaliculus may be due to a scar or a foreign body. Of the latter an eyelash is the commonest, a "concretion" less frequent. An eyelash usually projects somewhat from the punctum and is easily removed with forceps. Concretions are masses of the mycelium of a fungus, usually actinomyces. They are removed by dilating the canaliculus, slitting it, curetting it and injecting a solution of penicillin.

Congenital anomalies of the puncta and canaliculi are occasionally seen. The puncta may be absent or constricted; there may be two puncta in a lid, both generally opening into the same canaliculus. Sometimes a groove is found instead of a canaliculus.

Dacryocystitis or inflammation of the lacrimal sac is not uncommon. It may occur in an acute or chronic form.

Chronic dacryocystitis is the more common. The essential symptom is epiphora, aggravated by such conditions as exposure to wind. There may be a swelling at the site of the sac (a *mucocoele*) and the caruncle and neighbouring parts of the conjunctiva are frequently inflamed. On pressure over the sac, muco-pus or pus regurgitates through the puncta, or more rarely passes down into the nose. Bacteriological examination of the fluid demonstrates the presence of an extraordinary number of bacteria—staphylococci, pneumococci, streptococci, etc.; of these the pneumococcus is sometimes present in virulent form. This fact is of considerable importance since it explains the frequency with which hypopyon ulcer arises in these cases and the danger of panophthalmitis if any intra-ocular operation is undertaken. Dacryocystitis is a constant menace to the eye since minute abrasions of the cornea are of almost daily occurrence and such an abrasion is liable at any moment to become infected and give rise to an ulcer.

Chronic dacryocystitis is commonly attributed to the effects of stricture of the nasal duct arising from chronic inflammation, usually of nasal origin. Obstruction to the lower end of the nasal duct may be caused by the pressure of nasal polypi, a hypertrophied inferior turbinate bone, extreme deviation of the septum, and so on.

Untreated chronic dacryocystitis never undergoes spontaneous resolution. The condition tends to progress and the walls of the sac ultimately become atonic, the contents never being evacuated except by external pressure. At any time an acute inflammation may arise leading to the formation of a lacrimal abscess. This sequel may be caused by injudicious treatment by probing which

may result in an abrasion of the epithelial lining through which the pericystic tissues may become infected.

Dacryocystitis may occur in the new-born ; in these cases it is generally due to imperfect canalization of the epithelial cord in which the nasal duct is formed. Occasionally dacryocystitis in babies is very intractable : such cases are often tuberculous or syphilitic, usually originating in caries of the surrounding bones ; such an ætiology is rare in adults.

Treatment. In the new-born, penicillin drops should be ordered and minute directions should be given for expressing the contents of the sac, a manœuvre which should be done very frequently. Many cases will be cured by this treatment. If, however, a few weeks elapse without marked improvement, an anæsthetic should be given and *probing of the naso-lacrimal duct* performed, the greatest care being exercised to avoid injuring the walls of the duct ; it is unnecessary to slit the canaliculus.

The punctum and canaliculus are dilated with a Nettleship's dilator and a small probe (No. 1 or 2) is inserted vertically downwards into the canaliculus, then passed gently but firmly inwards until the point is felt against the lacrimal bone. The probe is then rotated upwards and towards the middle line, and pushed down the nasal duct until it touches the floor of the nose ; it should be remembered that the duct is short in the new-born. Little force is required if rightly applied in the line of the duct (p. 506). Since much harm may be done by unskilled probing, these cases should be treated by an expert. The passage of a probe *once* will cure most congenital cases.

In adults, repeated *syringing of the naso-lacrimal duct* may first be attempted particularly in recent cases with a view to reducing the swelling of the inflamed mucosa and restoring patency. The conjunctival sac is anæsthetized, the punctum dilated and the sac syringed out with a lacrimal syringe (Fig. 430). The point of the cannula being inserted into the canaliculus, two or three syringefuls of penicillin solution (10,000 units per ml.) are passed ; probably the whole of the fluid will regurgitate through the upper canaliculus. The operation should be repeated daily and in many cases the fluid will pass freely down into the nose in a few days ; when this occurs the syringing should be repeated at increasing intervals. A number of cases can be cured in this manner particularly if the patient is told to squeeze out the contents of the sac frequently in the intervals between syringing.

In the meantime the condition of the nasal passages should be thoroughly investigated by an expert, and any pathological condition likely to cause inflammation or obstruction of the nasal duct treated. The condition of the lacrimal passages may also be visualized radiologically after injecting lipiodol with a lacrimal syringe into the punctum and the upper part of the sac ; in this way the site of an obstruction may be located.

If the case is still recalcitrant, either excision of the lacrimal sac or the establishment of permanent drainage into the nose by dacryocystorhinostomy must be undertaken. The latter operation, properly performed, removes the disease and retains the function of drainage.

Many surgeons treat chronic dacryocystitis by repeated probing, probes of increasing calibre being passed down the nasal duct into the nose (Fig. 429). The objections to this method of treatment are—(1) it is impossible to probe the swollen and inflamed duct without injuring the walls ; (2) such injury may lead to infection of the surrounding tissues and an acute cellulitis ; (3) healing of the abrasions is accompanied by the formation of connective tissue which contracts when it organizes and leads to a fibrous stricture instead of obstruction by swollen mucous membrane ; (4) probing is always painful, and when once begun has to be continued for a prolonged period ; (5) most cases are alleviated only temporarily, fresh courses of probing being required at intervals. Silver preparations should never be injected immediately after slitting the canaliculus or probing since their entry through an abrasion into the subcutaneous tissues may result in a violent orbital cellulitis and even atrophy of the optic nerve. An unsightly and permanent staining of the skin (*argyrosis*) may also follow.

Excision of the Lacrimal Sac (Dacryocystectomy) is performed as follows. The operation can be undertaken with local analgesia, the conjunctival sac being anæsthetized and the area surrounding the sac with Procaine and adrenaline. An injection is made through the skin just above the fundus of the sac and along the upper canaliculus ; a second along the lacrimal crest over the sac, carried deeply along the floor of the orbit where the sac joins the nasal duct ; and final injections are made into the skin of each lid, 3 mm. from the centre of the lid margin. The ipsilateral nasal fossa is sprayed with cocaine and adrenaline, and may be packed with ribbon gauze

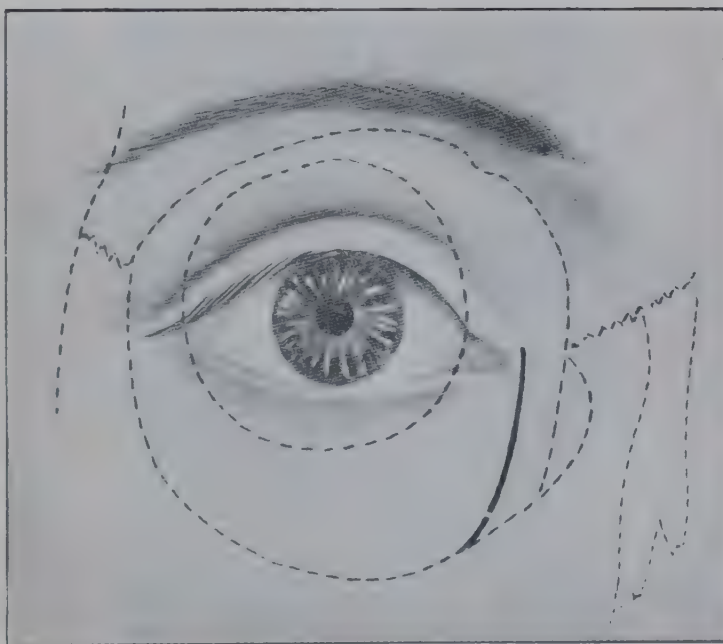


FIG. 433. Incision for excision of the lacrimal sac. The broken lines indicate the bones and orbital margin ; also the limits of the conjunctival sac.

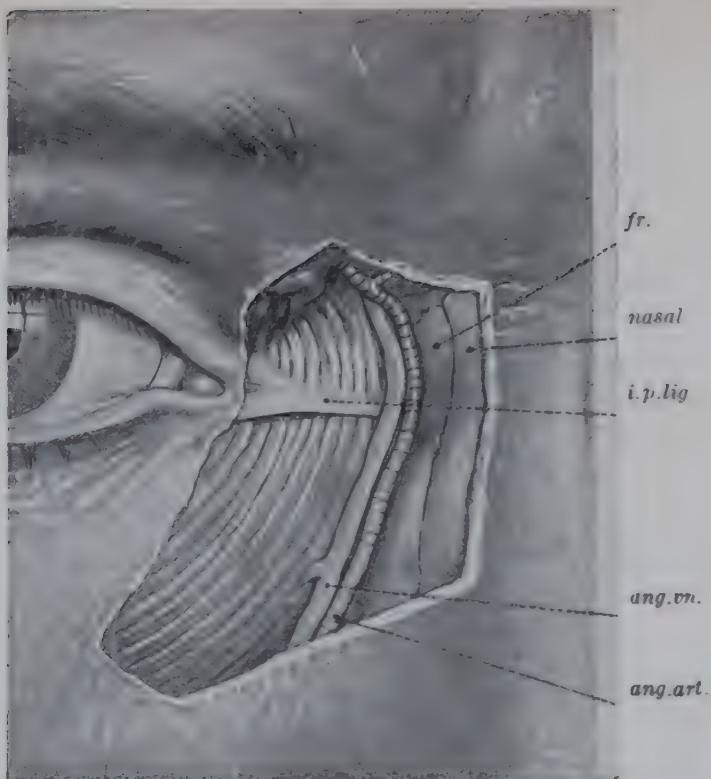


FIG. 434. Relations of angular artery and vein. *ang.art.*, angular artery; *ang.vn.*, angular vein; *i.p.lig.*, internal palpebral ligament; *nasal*, nasal bone; *fr.*, frontal process of superior maxilla. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

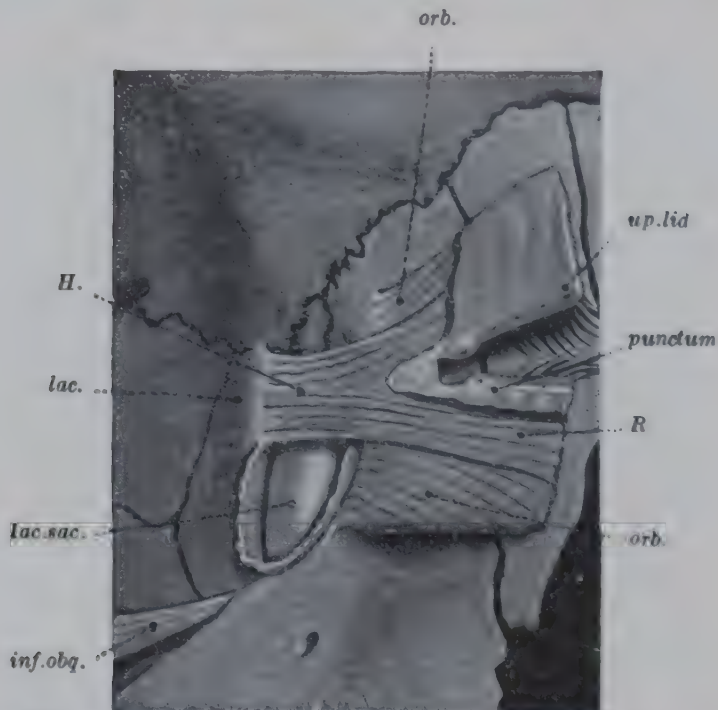


FIG. 435. Lacrimal sac. *H.*, Horner's muscle; *lac.*, lacrimal bone. *lac.sac.*, lacrimal sac; *inf.obq.*, inferior oblique; *orb.*, orbicularis. *R.*, Riolan's muscle. (Eugene Wolff. "Anatomy of the Eye and Orbit." Lewis, London.)

soaked in an oily solution of the same drugs. The canaliculi are then dilated, and the lacrimal sac irrigated with warm saline. Five minims of sterile melted wax impregnated with methylene blue may be injected so as to assist in the identification of the sac. The lids are temporarily closed with mattress sutures passed through the skin of the lid margins in order to avoid the danger of an infected abrasion.

With the skin stretched by moderate traction at the outer canthus, a curved incision is made, beginning 2 mm. above the medial palpebral ligament and 3 mm. to the nasal side of the inner canthus; it is carried vertically downwards for 4 mm., and then outwards along the line of the anterior lacrimal crest to a spot 2 mm. below the inferior orbital margin (Figs. 433-35). The skin of the temporal edge of the incision is undermined for 2 or 3 mm., but not that of the nasal edge owing to risk of wounding the angular vein or its branches. The orbicularis is split in the line of the incision, and a lacrimal retractor (Fig. 431) inserted so as to retract it with the skin. The lacrimal fascia is exposed and incised along the anterior lacrimal crest, thus bringing the bluish sac into view. This is freed from the bone on the nasal side by the blunt dissector (Fig. 432) and from its other connections by careful dissection until it remains attached only below to the nasal duct. The sac is drawn forwards and twisted two or three times in pressure forceps until it tears away from the duct. The upper end of the duct is curetted, and a probe passed down into the nose. The lids are now released and the epithelium lining the canaliculi removed by a canaliculus rasp. The orbicularis is sutured with catgut, and the skin incision by a continuous subcuticular suture. The eye is irrigated and an antibiotic instilled. A pyramid-shaped gauze dressing with its apex against the wound is firmly applied.

When the operation is satisfactorily performed there is no regurgitation on pressure over the scar. If after a week or two there is still some regurgitation, it suggests that part of the mucous membrane, usually the fundus of the sac, has been left behind, and the operation must be repeated. The remnant usually lies above the palpebral ligament and it is therefore advisable to divide the ligament in some cases to obtain a good view. Sometimes regurgitation is due to leaving in place the mucous membrane of the upper part of the nasal duct; this will not occur if the duct is well curetted.

Epiphora usually persists for a time after excision of the sac but gradually wears off, since, as already stated (p. 507), in normal conditions the tears evaporate from the surface of the globe. An unavoidable epiphora will, however, still occur on exposure to wind. If the post-operative epiphora persists it is usually due to the chronic conjunctivitis set up by the mucocele and this should be treated by astringent lotions; it is not necessary to remove the lacrimal gland, as has been advocated.

Dacryocystorhinostomy is a more difficult operation. The early steps are the same as for excision of the sac. An opening is then made through the lacrimal bone into the nose; the nasal mucosa is freed and the lacrimal sac incised so as to fashion two panels (Fig. 436). Fig. 437 shows the suturing of the posterior panels. The anterior panels are similarly sutured, thus covering the bony aperture with mucous membrane. When successful this operation restores a quasi-normal flow of the tears into the nose, but unless the opening is large and local infection has been controlled, the opening from the sac through the bone into the nose may become blocked with granulations or closed by scar tissue.

We have already noted the danger of performing an intra-ocular operation in the presence of lacrimal infection. Preliminary

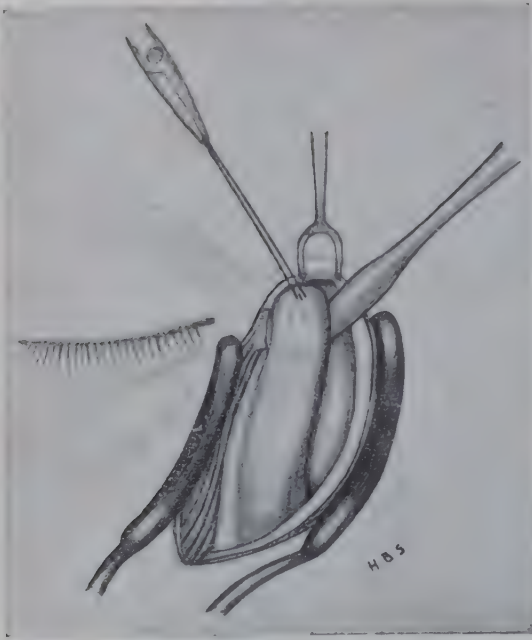


FIG. 436.

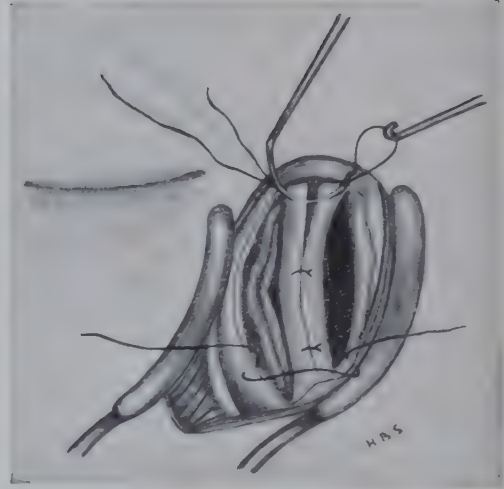


FIG. 437.

surgery on the sac is the safest treatment, but in cases of urgency the sac must be completely isolated from the conjunctival sac. This may be effected by syringing with an antibiotic and cauterizing the punctum by a diathermy needle, thus sealing it up with a cicatrix. This procedure may be followed by the development of a lacrimal abscess, but in the meantime the wound in the globe has probably healed, and in any case the pus is evacuated through the skin and not into the conjunctival sac.

Acute Dacryocystitis. *Lacrimal abscess* may be due to acute inflammation of the sac or to suppuration starting in the peri-



FIG. 438. Acute dacryocystitis.



FIG. 439. Lacrimal fistula.

cystic tissues. The skin over the sac becomes red and swollen. The redness and swelling rapidly extend to the lower lid and upper part of the cheek, so that the condition may be easily mistaken for erysipelas. There is severe pain, and often some fever. The abscess usually points below and to the outer side of the sac owing to gravitation of the pus to the margin of the orbit. If it opens spontaneously pus continues to be discharged for some time, and a permanent fistula is likely to result (Figs. 438-9).

Treatment. General treatment by antibiotic drugs should be instituted at once (Chapter 14). If seen at the beginning of the process an attempt may be made to draw the contents of the sac into the nose by cocainizing the ipsilateral nasal fossa and inserting a tampon soaked in adrenaline (1 in 2,000) over the opening of the naso-lacrimal duct. An injection of 5 minims of adrenaline (1 in 2,000) is made into the lacrimal sac. In some early cases the muco-pus can then be coaxed down the nasal duct.

Hot bathing should be persevered with and the abscess should not be opened unless it is pointing under the skin, in which case it should be opened by a small incision, the pus gently squeezed out, a piece of rubber-glove drain inserted, and a dressing of penicillin and sulphonamide powder applied.

If the discharge continues for a long period the cavity should be well curetted and again drained. Sometimes the epithelial lining of the sac is destroyed by the purulent inflammation, the sac is permanently sclerosed, and resolution is complete. In other cases some of the mucous membrane escapes destruction and a fistula may follow; the sac should then be re-opened along a director introduced through the fistula and the remnants extirpated. This, however, should not be done until several weeks after the acute inflammation has subsided.

Stricture of the nasal duct has already been referred to incidentally. It is probable that many intractable fibrous strictures are caused by probing; others arise spontaneously as the result of destruction of the epithelium by extension of inflammation from the nose or lacrimal sac. Occasionally bony strictures occur, usually caused by a fractured maxilla, inflammation of the antrum, or caries.

Treatment. A common treatment of stricture of the nasal duct used to be dilatation with probes. The objections to this treatment have been mentioned. A nasal drainage operation (p. 515) should be performed in these cases.

CHAPTER 33

DISEASES OF THE ORBIT

It is unnecessary to describe the anatomy of the orbit and its contents here. The student is recommended to revise his knowledge of the subject, paying special attention to the relations of the nasal cavities and their accessory sinuses, and to the communications with the interior of the cranial cavity by way of the optic foramen and

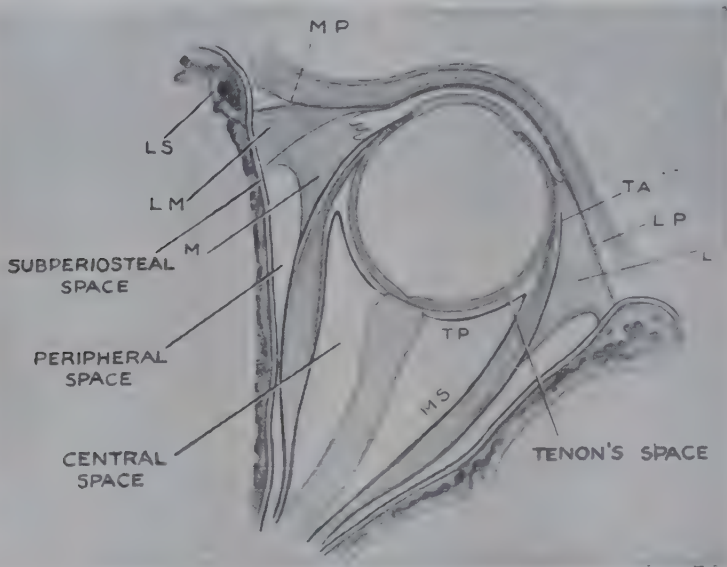


Fig. 440. Horizontal section through the orbit. L, lateral check ligament ; LM, lacrimal portion of orbicularis muscle ; LP, lateral palpebral ligament ; LS, lacrimal sac ; M, medial check ligament ; MP, medial palpebral ligament dividing into a superficial and a deep band ; MS, muscle sheaths ; TA, anterior part of Tenon's capsule ; TP, posterior part of Tenon's capsule.

sphenoid fissure. The intimate adhesion of the dural sheath of the optic nerve to the walls of the optic foramen is of great pathological importance, and the relations of the intra-orbital to the intracranial circulation must be thoroughly appreciated. The eye is slung in position in the orbit by fascia, one sheet of which, Tenon's capsule, forms a socket in which the globe moves. This, with the sclera, forms a lymphatic space, lined completely with endothelium. The extrinsic muscles of the eye do not perforate this capsule, but invaginate it, the fascia being reflected from their surfaces.

From the surgical point of view there are thus four spaces which are relatively self-contained, within each of which inflammatory processes are contained for a considerable time, and each of which

must, if necessary, be opened separately (Fig. 440) : (1) The *subperiosteal space* between the bones of the orbital wall and the periorbita ; (2) the *peripheral orbital space* between the periorbita and the extra-ocular muscles which are joined by fascial connections making a more or less continuous circular septum ; (3) the *central space*, a cone-shaped area enclosed by the muscles (the "muscle cone") ; and (4) *Tenon's space* around the globe.

The position of the eye in the orbit is important. The normal position of the eye is such that a straight-edge applied vertically to the middle of the upper and lower margins of the orbit just touches the closed lids over the apex of the cornea. There are individual variations which are of no pathological importance when symmetrical ; in all cases of doubt the two sides should be compared.

Accurate estimates of the amount can be obtained only by special mechanical devices (exophthalmometers) ; a simple one is illustrated in Fig. 441. A convenient test is the following : the patient is seated, the surgeon standing

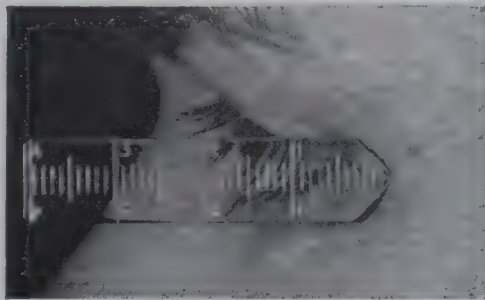


FIG. 441. A simple exophthalmometer. The instrument is made of a transparent plastic with a groove which fits into the outer bony margin of the orbit. The scale is engraved on both sides of the solid bar of plastic and the observing eye is aligned to read off the level of the apex of the cornea so that the scales on either side of the bar and the apex of the cornea are in one straight line. In the illustration, the two scales at 3 cm. are seen out of alignment, and at 4 cm. this tendency is increased.

behind him. The surgeon holds the patient's head in such a manner that he looks straight down the nose. He then rotates the head backwards until he can just see the apex of one cornea. If he can see more of the other cornea, that eye is relatively proptosed.

Abnormal protrusion of the globe is called *exophthalmos* or *proptosis*. It is much commoner than abnormal retraction or *enophthalmos*. The former condition is due to many causes, among which increase in the orbital contents is the most important. Slight prominence of the eyes accompanies high myopia, paralysis of the extrinsic muscles, stimulation of Müller's muscle by cocaine, and occurs as an idiosyncrasy, especially in very obese people. Unilateral proptosis occurs in orbital cellulitis from any cause, thrombosis of the orbital veins with or without implication of the cavernous sinus,

arterio-venous aneurysm, tumours of the orbit and its contents, and orbital hæmorrhage or emphysema. Bilateral exophthalmos occurs in endocrine exophthalmos, bilateral proptosis in the later stages of thrombosis of the cavernous sinus, empyema of the accessory sinuses of the nose, symmetrical orbital tumours (lymphoma, pseudo-leucæmia), and as a result of diminished orbital volume in oxycephaly or "tower-skull" and leontiasis ossea.

Enophthalmos is generally due to severe injury in which the bones of the floor of the orbit are fractured so that the globe falls downwards and backwards, or to orbital cellulitis followed by mechanical retraction by fibrous tissue.

ORBITAL INFLAMMATION

Periostitis is not common but particularly affects the orbital margin (Fig. 442). It is most often due to injuries, extension of in-



FIG. 442. Periostitis and osteomyelitis of the orbital roof (R. C. Gamble).

flammation from neighbouring parts, tuberculosis or syphilis. Tuberculous periostitis is most frequent in children, syphilitic in adults: in the former, caries of the bones results; the latter is gummatous. In traumatic cases the margin is naturally most affected, but a traumatic element is often an exciting cause in the other cases, so that in them also the margin most frequently suffers.

When situated at the margin, the inflamed part is swollen and tender; the swelling is intimately connected with the bone, so that it cannot be moved over it. If untreated the disease tends to progress to suppuration; an abscess is formed, and when it discharges or is opened, rough bone can be felt with a probe. In tuberculous cases particularly a fistula may result, the edges of the aperture being bound down to the bone so that a depressed cicatrix is formed. The fistula remains open until all the necrosed bone is extruded. The cicatrization may lead to displacement of the lid—ectropion, lagophthalmos, and so on.

Periostitis of the deeper parts of the orbit causes less definite signs. There is more pain of a deep-seated character. There may be proptosis with deviation of the direction of the eye. Often the true nature of the disease is only discovered by an exploratory operation, or by the evacuation of pus, for the case may present all the features of orbital cellulitis (*q.v.*). If the roof of the orbit is involved, the pus may discharge into the cranial cavity, life being endangered by meningitis or cerebral abscess. If the apex of the orbit is implicated, various ocular motor palsies may develop together

with trigeminal anæsthesia and neuralgia, and occasionally amaurosis due to involvement of the optic nerve (the *syndrome of the apex of the orbit*). In the case of a gumma the roof of the orbit is generally involved, the deviation of the eye is downwards, and there is rapid loss of movement owing to involvement of the extrinsic muscles together with severe supra-orbital neuralgia which is worse at night.

Treatment is determined by the ætiological factor. In most cases general treatment by antibiotic drugs to which the infecting organism is susceptible has a dramatic effect. If suppuration supervenes, the abscess is opened and any carious bone removed, hot fomentations being applied previously if necessary.

In deep-seated periostitis an exploratory operation may be necessary and should not be too delayed. An incision is made through the skin at the margin of the orbit at a site determined by the signs, the periorbita being incised to open the subperiobital space. Sinus forceps are passed in, and opened, the greatest care being exercised to keep closely to the bony walls and avoid unnecessary damage to the orbital contents. Special care must be taken not to injure the pulley of the superior oblique. If pus is found, a small drainage tube or a strip of rubber glove or cyanide gauze is inserted. In periostitis of the inner wall, the bone may be widely diseased and extensive operations, involving the opening of the frontal or ethmoid sinuses, may be essential, with or without drainage through the nose ; these cases often do well. Exploration of the orbit in children is more difficult than in adults since the eye is relatively larger in comparison with the size of the orbit, leaving little room between the globe and the orbital wall. In rare cases it may be advisable to perform Krönlein's operation (*q.v.*).

Orbital Cellulitis is purulent inflammation of the cellular tissue of the orbit. It is due most frequently to extension of inflammation from neighbouring parts, especially the nasal sinuses, occasionally (particularly in infants) the teeth ; other less common causes are deep injuries, especially those with a retained foreign body, septic operations, particularly enucleation of the eyeball, facial erysipelas, or metastases in pyæmia.

There is great swelling of the lids, with chemosis. The eye is proptosed, and its mobility impaired with resulting diplopia. Pain is severe, increased by movement of the eye or pressure. Fever is present, and cerebral symptoms may arise. Vision may not be affected, or it may be reduced owing to retrobulbar neuritis. The fundus is difficult to examine ; it may be normal or show engorgement of the veins and optic neuritis, passing later into optic atrophy. An abscess is formed which usually points somewhere in the skin of the lids near the orbital margin, or it may empty into the conjunctival fornix. Panophthalmitis may supervene and there is grave danger of extension to the meninges and brain as purulent meningitis, cerebral abscess or thrombosis of the cavernous sinus.

Treatment. General treatment with antibiotic drugs should be instituted at once (Chapter 14), and if instituted reasonably early, resolution may rapidly follow. Hot bathings and medical diathermy, if available, may be applied, but must not be relied upon too long. An early incision as in orbital periostitis (*q.v.*) is made and the peripheral surgical space opened. If pus is not reached multiple incisions within the orbit should not be attempted, since the tension is already relieved and a track is prepared for its evacuation. If the source of infection is obscure, the nose and other likely seats must be investigated, and the primary focus treated.

Thrombosis of the Cavernous Sinus may be due to extension of thrombosis from various sources.

The anatomy of the venous channels which communicate with the cavernous sinus is of great importance in this connection (Figs. 443, 444). The superior and inferior ophthalmic veins enter it in front and



FIG. 443. Tributaries of the cavernous sinus (lateral view).
(For explanation of lettering, see Fig. 444.)

the superior and inferior petrosal sinuses leave it behind. It communicates directly with the pterygoid plexus through the middle meningeal veins and the veins of Vesalius, and indirectly through a communicating vein from the inferior ophthalmic to the pterygoid plexus. The anastomoses of the ophthalmic veins with the frontal and angular open up a communication with the face. Labyrinthine veins opening into the inferior petrosal sinus afford a communication with the middle ear. Numerous tributaries throw it into direct or indirect communication with most parts of the cerebrum. The mastoid emissary vein places the sinus in communication with the subcutaneous tissues behind the ear through the lateral sinus and superior petrosal sinus; it is this communication which is of great diagnostic importance, since swelling behind the ear may decide the question of thrombosis in each direction along these sinuses. The sinus of one side communicates with that of the other by two (or sometimes three) transverse sinuses which surround the pituitary body.

Infection may occur *via* the orbital veins, as in erysipelas, septic wounds of the face, orbital cellulitis, and infective conditions of the

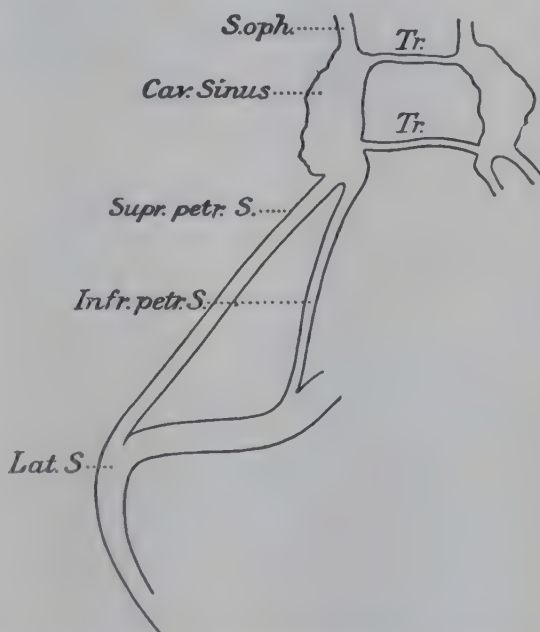
mouth, pharynx, ear, nose and accessory sinuses, or as a metastasis in infectious diseases or septic conditions. On more than one occasion the tragedy of bilateral blindness has resulted from an event so simple as the injudicious squeezing of a furuncle on the nose.

The patient presents almost the same symptoms and signs as in orbital cellulitis. If in addition there is œdema in the mastoid region behind the ear the diagnosis is certain, for this is due to thrombosis of the emissary vein. A further point of diagnostic importance is transference of the symptoms to the opposite eye, which occurs in 50 per cent. of cases, whereas bilateral orbital cellulitis is very rare. The first sign is often paralysis of the opposite lateral rectus, and this should be carefully watched for in any suspicious case of in-

FIG. 444. Tributaries of the cavernous sinus (from above).

Explanation of Figs. 443, 444.

Ang., angular vein. *Cav.* Sinus, cavernous sinus. *Com.*, communicating vein. *Fac.*, facial vein. *Fr.*, frontal vein. *I. oph.*, inferior ophthalmic vein. *I. petr.* (*Infr. petr. S.*), inferior petrosal sinus. *Jug. V.*, jugular vein. *Lab.*, labyrinthine veins. *Lat. Sinus. (S.)*, lateral sinus. *Mas. Em.*, mastoid emissary vein. *Mid. Men.*, middle meningeal veins. *Nasal*, nasal veins. *Pt. px.*, pterygoid plexus. *S.o. (oph.)*, superior ophthalmic vein. *Supra-orb.*, supra-orbital vein. *S. (Supr.) petr.*, superior petrosal sinus. *Tr.* transverse sinus.



flammatory unilateral proptosis. It must be remembered, however, that thrombosis of the sinus may be a complication of cellulitis.

There is severe supra-orbital pain owing to implication of the branches of the ophthalmic division of the fifth nerve, and paresis of the ocular motor nerves. In the later stages the eye is immobile, the pupil dilated, and the cornea anæsthetic. Proptosis occurs in nearly all cases, but is of late onset in those of otitic origin.

It is commonly stated that the retinal veins are greatly engorged, but in many cases this is certainly not true. When this occurs it is usually accompanied by pronounced papillitis, and both signs indicate extensive implication of the orbital veins and tissues. Simultaneous thrombosis of both cavernous sinuses, with proptosis and papillitis, occurs in diseases of the sphenoid sinuses. Typical papilloœdema is commonest in otitic cases and indicates meningitis or cerebral abscess: it is bilateral, but more pronounced on the side of the aural lesion.

Thrombosis of the cavernous sinus is accompanied by rigors, vomiting and severe cerebral symptoms. Before the advent of the modern antibiotic drugs the patient usually died, but their early use in massive (intravenous) doses together with anti-coagulants may bring about resolution.

Tenonitis is inflammation of Tenon's capsule: it may be serous or purulent. There is proptosis, the globe being pushed straight forwards with limitation of movement and pain on any attempt to move the eyes. There may be some œdema of the lids, and chemosis. It may occur in severe iridocyclitis, and is constant in panophthalmitis. Simple serous tenonitis is rare, and has been attributed to influenza, gout or rheumatism.

Treatment consists in general treatment by the antibiotic drugs (Chapter 14) together with the application of hot bathings and the evacuation of pus, if it forms. When it occurs in association with panophthalmitis, it requires no special treatment.

DISTENSION OF THE ACCESSORY SINUSES OF THE NOSE

The accessory sinuses of the nose—the frontal, ethmoid and sphenoid sinuses, and the antrum of the superior maxilla—are separated from the orbit only by thin plates of bone. The orifices which form the communication between these cavities and the nose are liable to become occluded by catarrh, polypi or neoplasms, so that the normal sero-mucous discharge is unable to drain into the nose; the cavities therefore become distended with fluid, and if pyogenic organisms are present, pus may be formed. The treatment of the conditions thus set up cannot be considered part of the functions of the ophthalmic surgeon, but he must be prepared to diagnose them since they not infrequently appear for the first time in the ophthalmic clinic. This is particularly the case in distension of the frontal, ethmoid and sphenoid sinuses. Of these the frontal sinus suffers most often.

Distension or empyema of the frontal sinus causes bulging at the upper and inner part of the orbit (Fig. 445). There may be some proptosis and displacement of the eyeball downwards and outwards, œdema of the upper lid or slight ptosis. There is considerable pain and tenderness with severe headache.

Distension of the ethmoid cells by polypi, new growths or inflammatory products may also cause bulging into the orbit and displacement of the globe. Diplopia, chemosis, venous engorgement and ptosis may be caused.

In all cases there is often discharge from the nostril of the same side, or manifest disease of the nasal cavities. Owing to erosion of the walls of the sinus the fluid may extend under the periorbita causing bulging into the posterior part of the orbit or orbital cellulitis. Occasionally retrobulbar neuritis may occur, a complication

most probable with inflammation and distension of the sphenoid cells which lie in close proximity to the optic nerve.

In doubtful cases help may be afforded by a skiagram. *Treatment*, apart from general antibiotic therapy, is essentially nasal, but an opening into the orbit may be necessary if cellulitis develops (*q.v.*)



FIG. 445. Fronto-ethmoidocele (M. M. Kulvin).

TUMOURS OF THE ORBIT

Orbital tumours are rare. **Benign growths** include dermoid cyst, dermo-lipoma (p. 183), angioma, osteoma (Fig. 446), plexiform neuroma (p. 502), meningioma and meningo-encephalocele.

Dermoid cysts appear as swellings under the lid, usually at the upper and outer angle; they contain sebaceous material derived from sebaceous glands in the walls, which are lined with epithelium and possess hair follicles; they sometimes contain foetal remnants (teratoid cysts).

Clinically they may be mistaken for *meningo-encephaloceles* protrusions of the cerebral contents, which usually occur at the upper and inner angle where there are most sutures between bones. In the latter (1) the tumour is immovably attached to the bones; (2) the



FIG. 446. Orbital osteoma (Tweedy).

hole in the bone may be palpable; (3) pulsation, synchronous with respiration and the pulse, increasing in amplitude on straining, can be seen; (4) pressure may cause diminution in size due to fluid being pressed back into the cranium; (5) exploratory puncture (which should only be undertaken with full aseptic precautions) produces clear fluid with the characteristics of cerebro-spinal fluid.

Osteomata start from the nasal sinuses, usually from the frontal (Fig. 446) ; they are intensely hard and often large, producing great displacement of the globe.

Meningiomata, apart from those originating in association with the optic nerve sheath (*vide infra*), generally arise in association with the intracranial meninges and invade the orbit secondarily causing a hyperostosis. The most typical are those arising from the lateral portion of the sphenoid ridge—slowly-growing tumours causing proptosis and visual failure by pressure on the optic nerve.

Hæmopoietic tumours may occur—the various types of reticular tumour (lymphoma, lymphosarcoma, reticulo-sarcoma, Hodgkin's disease, etc.) and chloroma. *Angiomata* are rare.

Malignant tumours of the orbit are usually sarcomata, although carcinomata derived from the lacrimal gland (p. 508) or by extension from the nasal mucous membrane also occur.

Primary tumours of the optic nerve manifest themselves clinically as orbital tumours. They are rare, and are found on anatomical examination to consist of two groups—neuromata and meningiomata. Of these the former are more numerous. Selective staining shows that they are nearly all true gliomata springing from

the neuroglia ; a few are endotheliomata or fibromata, in the proportions roughly of 20 : 4 : 1. They spread slowly within the sheaths, and death is due to intracerebral extension. *Meningiomata* are usually endothelial, springing from the arachnoid and endothelial lining of the dura ; they infiltrate the nerve and may spread to the orbit. The rare extradural growths springing from the dura are chiefly fibrous in structure (fibromata), and may fill the orbit (Fig. 447). All these optic nerve tumours are locally malignant but show little or no tendency to metastasis.

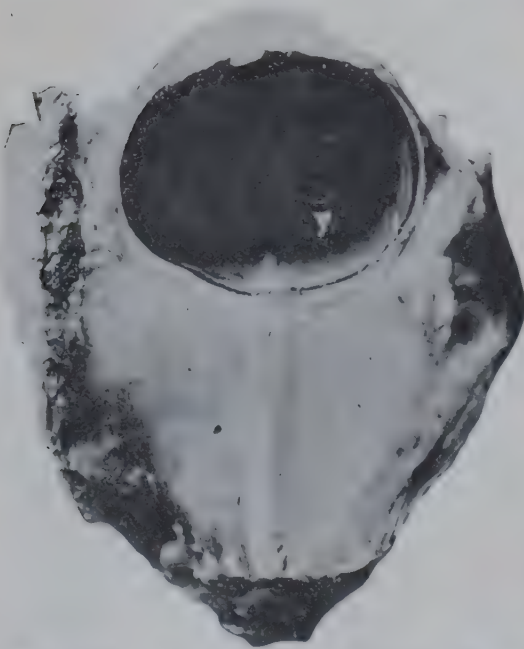


FIG. 447. Extradural tumour of the optic nerve ($\times 1.4$).

Secondary tumours also occur—carcinoma, hypernephroma, and in young children, sympathetic neuroblastoma ; the parent growth of the last is usually in the adrenal medulla.

Malignant nasopharyngeal tumours form 0.4 per cent. of all cases of cancer ; 38 per cent. of cases show ophthalmo-neurological

symptoms, these being the earliest signs in 16 per cent. of cases. The fifth and sixth nerves are most frequently involved ; more rarely the third, fourth and the optic nerve. Quadrantic and hemianopic lesions are rare, thus distinguishing these cases from lesions in the neighbourhood of the sella turcica. The presence of abducens paralysis, especially if associated with impairment of vision, Horner's syndrome or proptosis or enlargement of the cervical lymph nodes, should suggest a naso-pharyngeal growth. Treatment is radiological.

The **lipodystrophies** may give rise to tumour-like formations resulting from the reaction of the reticulo-endothelial system to the deposition of lipids—*diabetic exophthalmic dysostosis* (Hand-Schüller-Christian disease), *xanthomatosis*, etc.

The *symptoms* of most orbital tumours are similar ; the differential diagnosis must frequently be made by biopsy.

Most orbital tumours cause proptosis, which is very rarely straight forwards except in the case of optic nerve tumours ; this is an important diagnostic feature (Fig. 448). The proptosis is slowly



FIG. 448. Orbital tumour, showing proptosis and downward displacement of the globe.

progressive and (with the exception of the hæmopoietic tumours and sympathetic neuroblastomata) usually unilateral. The mobility of the eyeball is impaired in the direction towards the position of the tumour ; diplopia is therefore common, while papilloedema may be present, especially with optic nerve tumours. Optic atrophy from pressure on the nerve occurs in the other forms. The tumour may be palpable by the finger inserted between the globe and the orbital wall. The lymphatic nodes are seldom affected.

Careful examination of neighbouring parts—nose, antrum, mouth and especially the nasopharynx—must be made to determine whether the invasion of the orbit is secondary or whether the growth is primarily orbital. In all cases an X-ray examination should be made.

Treatment. An exploratory operation and removal of a portion of the growth for microscopic examination may be a necessary preliminary to radical treatment. It may be feasible to remove dermoid cysts and some other benign tumours without injury to the globe, although its mobility is likely to be impaired in extensive operations. As already mentioned, many malignant orbital growths

show little tendency to metastasis, so that their treatment may be more conservative than is usual in other parts of the body. Some tumours (particularly the reticular tumours) respond to irradiational treatment and recurrences in the orbit or metastases should be treated by these means.

The majority, however, requires surgical excision. Three routes of approach with retention of the eye are available—an anterior orbitotomy, wherein an incision as for cellulitis (*q.v.*), made anteriorly at the orbital margin, provides access to the anterior half of the orbit; a lateral orbitotomy provides access to the deeper parts of the orbit and is a valuable exploratory procedure; while the apex of the orbit is best reached through a trans-frontal (intracranial) approach. In the case of more malignant types of tumour their complete removal is imperative at all costs, and the eye, even if normal, may have to be sacrificed. In these cases, as well as in recurrence or in orbital extension of malignant intra-ocular growths (retinoblastoma, malignant melanoma of the uveal tract), it may be necessary to remove the whole contents of the orbit by exenteration.

In *lateral orbitotomy* (*Krönlein's operation*) a semilunar incision is made vertically just outside the outer canthus, the convexity being directed forwards. The bone is chiselled through at the upper and lower outer angles of the orbit, and bone, muscle and skin are reflected backwards in one flap. The part of the orbit immediately posterior to the globe is thus freely exposed.

In *exenteration of the orbit* the lids may be retained if they are not implicated in the growth, but the free margins, carrying the cilia, should always be removed. If this is not done the lashes are troublesome when the lids become retracted into the orbit, as invariably follows. If the lids are removed the incision is carried through the skin at the margin of the orbit in its whole circumference. The orbital contents are separated from the walls by a periosteal elevator, so that they remain attached only at the apex of the orbit. The pedicle is then severed with strong scissors, or preferably by diathermy, thus avoiding hæmorrhage. At a later stage it may be advisable to apply split-skin grafts to the walls, since the lids and conjunctiva never afford sufficient epithelial covering, and the extension of the epithelium over so large a surface is a tedious process.

Endocrine Exophthalmos is a puzzling condition the ætiology of which is not yet understood. Two types occur—a mild exophthalmos associated with thyrotoxicosis (exophthalmic goitre) and an extreme exophthalmos occurring in any state of thyroid activity, but usually in hypothyroidism, often after a thyroidectomy.

The cause of exophthalmos in either type is obscure. That of thyrotoxicosis probably depends, in part at any rate, on fatty infiltration of the ocular muscles, while the associated retraction of the lid may be aided by contraction of Müller's muscle owing to the sensitizing action of thyroxine on sympathomimetic substances. The exophthalmos of exophthalmic ophthalmoplegia is due to œdema, lymphocytic infiltration and fibrosis of the orbital contents, particularly the extra-ocular muscles. These changes are probably due to a generalized disturbance of the endocrine system, possibly associated with the thyrotropic

hormone secreted by the anterior lobe of the pituitary gland which normally stimulates the thyroid gland to activity.

Exophthalmic Goitre (*Thyrotoxic exophthalmos*, *Graves's disease* or *Basedow's disease*) includes in its symptomatology in addition to exophthalmos all the signs of thyrotoxicosis—tachycardia, muscular tremors and a raised basal metabolism. From the ocular point of view, the exophthalmos in the early stages may be unilateral but usually becomes bilateral. There is a peculiar stare with retraction of the upper eyelid, so that there is an unnatural degree of separation between the margins of the two lids (Dalrymple's sign) (Fig. 449).



FIG. 449. Thyrotoxicosis, showing retraction of the upper lid and proptosis.

Normally, when the eye is directed downwards, the upper lid moves concordantly with it. In this disease the upper lid follows tardily or not at all (von Graefe's sign): this symptom is not always present and may occur in other forms of exophthalmos. There is diminished frequency of blinking and imperfect closure of the lids during the act (Stellwag's sign). There may be imperfect power of convergence (Möbius's sign), and often the skin of the eyelids shows pigmentation. Ophthalmoscopically, veins and arteries may be somewhat distended, but specific signs are absent. One or more of the cardinal symptoms may be absent. Recovery follows control of the thyrotoxic condition.

Exophthalmic Ophthalmoplegia (*Thyrotropic Exophthalmos*) usually commences in middle age with insidious signs of proptosis and external ophthalmoplegia typically asymmetrically divided between the two eyes and involving preferentially limitation of upward movement. The ocular muscles are enormously swollen, pale, oedematous and infiltrated, giving rise to an irreducible exophthalmos which may easily result in the development of an exposure keratitis or even dislocation of the globe (Fig. 450). The disease runs a self-limited course characterized by intermissions and relapses, more or less unaffected by any kind of treatment, but eventually tends to spontaneous resolution which, however, is rarely complete.

From the ophthalmological point of view the protection of the

exposed cornea is of paramount importance ; in less severe cases this is achieved by tarsorrhaphy (*q.v.*) ; in more severe cases a decompression of the orbit either by a lateral orbitotomy or transfrontally may be necessary.



FIG. 450. Exophthalmic ophthalmoplegia.

Pulsating Exophthalmos is generally due to arterio-venous aneurysm, the communication taking place between the internal carotid artery and the cavernous sinus. The eyeball is protruded and the blood vessels of the conjunctiva and lids are widely dilated. The angular vein and its branches near the inner canthus are very prominent, and they can be seen, or more easily felt, to pulsate synchronously with the arterial pulse since, owing to the arterio-venous communication, they are under arterial pressure. The patient complains of continual rumbling, as of a waterfall, and this can be heard on auscultation over the eye or orbit by the surgeon. The proptosis is diminished by steady pressure on the globe, and may be diminished or abolished by pressure on the common carotid artery of the same side, or sometimes only by pressure on the carotid of the opposite side also. Ophthalmoscopically, the veins of the retina are greatly distended : there may be papillœdema with defective vision, which may amount to complete blindness. There is often considerable pain from stretching of the branches of the fifth nerve.

The cause of the arterio-venous aneurysm is usually a severe blow or fall upon the head, and it is therefore commoner in men, but in most cases the walls of the artery are already degenerated. It may occur from arteriosclerotic or syphilitic disease without discoverable traumatism, especially when it occurs in women. The exophthalmos in rare cases subsides spontaneously. More commonly it increases, and may end in hæmorrhage or death from cerebral causes.

Treatment. If continuous pressure applied to the carotid artery stops the pulsation, ligation of the carotid may effect a cure, but recurrence of pulsation not infrequently occurs. Ligation of both internal and external carotid does not appear to give better results. The opposite carotid may also be tied, but this should not be done for some weeks after the first operation, owing to the risk of cerebral anæmia. This procedure also may fail to relieve the

condition, and in these cases intracranial ligation proximally and distally to the aneurysm has been practised, an operation of considerable difficulty and danger.

Intermittent Proptosis occasionally occurs, generally when the head is depressed, enophthalmos not infrequently being present in the erect position. The proptosis is increased by pressure on the corresponding jugular vein. It is usually due to varicosity of the orbital veins and has been found to be caused by intracerebral arterio-venous communications.

INJURIES OF THE ORBIT

Injuries to the soft parts usually arise from penetration of a foreign body which may be retained; the lids and eyeball are frequently implicated. The signs depend upon the particular structures injured. In most cases there is considerable hæmorrhage; as the blood does not find a ready exit proptosis may result and extravasation of blood under the conjunctiva and into the lids is common. Paralysis of the extrinsic muscles may be due to direct injury or to damage to the motor nerves. The optic nerve may be injured or severed with resultant atrophy. Avulsion of the disc, with the formation of a traumatic "coloboma" or "conus" of the disc, may occur, even without rupture of the sheath of the nerve. The eyeball may be perforated or contused or dislocated outside the lids. Retained foreign bodies are liable to set up suppuration and orbital cellulitis (*q.v.*).

A blow in the orbital region without the penetration of a foreign body may lead to an intra-orbital hæmorrhage; this may occur from pressure with forceps at birth. It is to be noted that an orbital and subconjunctival hæmorrhage is a frequent sign of a fracture of the base of the skull. Dislocation of the globe forwards between the lids occurs most often when the blow is directed from the outer side where the orbital margin affords least protection; sight is not necessarily lost after such a dislocation. Insane patients have been known to enucleate their eyes by gouging them out with their fingers.

Injuries to the bone most commonly affect the margin of the orbit but deep fractures may be caused by penetrating wounds or by severe contusions. Fractures near the orbital rim are easy to diagnose from the unevenness of the margin, sensitiveness to pressure, and sometimes crepitation. Deeper fractures may give rise to *emphysema* which may cause proptosis but is usually most evident in the lids. It is due to communication of the subcutaneous tissues with the nasal air sinuses so that air is forced into the tissues on blowing the nose, sneezing, straining, or coughing. The diagnostic signs are the considerable swelling and the peculiar soft crepitation on palpation.

In fractures of the orbital floor the globe may sink backwards and be depressed with a resultant troublesome diplopia (*traumatic enophthalmos*) (Fig. 451). It is important that such fractures be accurately

reposed at an early stage since the correction of the diplopia is otherwise difficult, involving grafting operations to rebuild the orbital floor and raise the eye. Reposition is effected through the antrum.

Fractures of the base of the skull may involve one or both optic foramina, in which case the optic nerve may be injured or pulsating

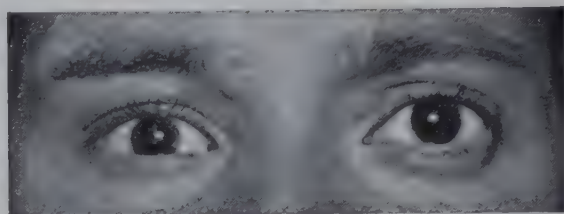


FIG. 451. Enophthalmos and downward displacement of the right eyeball due to a fracture of the floor of the orbit.

exophthalmos (*q.v.*) may ensue. Blindness without ophthalmoscopic signs may be caused in this manner: atrophy of the disc follows in three to six weeks.

Gunshot wounds of the orbit without direct involvement of the eye frequently produce concussion changes which appear ophthalmoscopically as coarse tracks of white exudate in the retina and choroid, large blot-like hæmorrhages and multiple small choroidal tears. These resolve into densely scarred areas fringed with pigment, with finer pigmentary disturbances elsewhere in the fundus (Plate XIX, Fig. 3). The site may give an indication of the direction of the track of the missile and assist in localizing a retained intracranial foreign body. Both eyes should be examined as the missile may have traversed both orbits.

Treatment. If there is a wound it must be cleansed and, if a foreign body is possibly retained, probed: it should be dusted with penicillin-sulphonamide powder and a prophylactic course of systemic antibiotic treatment given if indicated. Absorption of extravasated blood is often slow. The treatment of a retained foreign body depends upon its situation and the probability of subsequent infection. If the foreign body cannot be extracted with ease, a skiagram should be taken. If the position is such that serious manipulations would be necessary for its removal, and if there is evidence that the substance is aseptic, expectant treatment may be adopted. If suppuration occurs, the foreign body must be removed and the case treated as one of orbital cellulitis (*q.v.*).

SECTION VII

SYMPTOMATIC DISEASES OF THE EYE

CHAPTER 34

OCULAR MANIFESTATIONS OF DISEASES OF THE NERVOUS SYSTEM

THE ocular signs of nervous disease often appear superficially to be complicated and confusing but in most cases they are readily explained by the anatomy of the part of the nervous system involved. Even at the risk of some repetition the main ocular symptoms of these diseases will be summarized in this section without describing the diseases themselves.



FIG. 452. The common sites of aneurysms in the chiasmal region (Russell Brain).

CIRCULATORY DISORDERS

Intracranial aneurysms are not very rare. Those of ophthalmological interest affect the circle of Willis, where they are usually congenital but may be due to arterial degeneration or injury (Fig. 452). They give rise to ophthalmic symptoms in three ways :—

(1) By mechanical pressure on neighbouring structures in their slow growth, causing symptoms characteristic of a tumour in the chiasmal region (*q.v.*).

(2) By sudden increments in size and periodic slight leakages. An acute attack of severe pain develops, usually referred to the temple, associated with intermittent attacks of incomplete ophthalmoplegia involving ptosis and diplopia and visual impairment.

(3) By sudden bursting producing an apoplectic attack associated with *subarachnoid hæmorrhage*. There is sudden acute headache with vomiting and dizziness. Coma may rapidly supervene. If the patient survives, the ocular signs are palsies, especially of the IIIrd or VIth nerve; moderate papilloedema; retinal hæmorrhages, usually multiple in the neighbourhood of the disc, rather large, often subhyaloid, and occasionally spreading into the vitreous; proptosis; and defects in the visual fields. There is always blood in the cerebrospinal fluid, as shown by lumbar puncture.

Cerebral Hæmorrhage and Thrombosis. In the *occipital cortex* the posterior cerebral artery is usually involved; it supplies most of the occipital cortex and much of the temporal lobe. A lesion of this vessel thus causes a crossed homonymous hemianopia often with disturbances of the visuo-psychic areas (Fig. 269); the macular area is usually spared owing to overlapping with the middle cerebral artery at the posterior pole (p. 352). Obstruction of the middle cerebral artery produces visual agnosia with a crossed homonymous field defect affecting preferentially the upper quadrants of the field by involvement of the optic radiations.

A lesion of the *frontal motor cortex* (Fig. 365) causes a conjugate deviation of the eyes away from the side of the lesion in the irritative stage, which is reversed in the paretic stage. A hæmorrhage at the *internal capsule* produces a conjugate deviation of the head and eyes towards the side of the lesion with a contralateral hemiplegia. A hæmorrhage into the *pons* (below the decussation of the corticofugal fibres) produces conjugate deviation of the head and eyes away from the side of the lesion, that is, towards the hemiplegic side, and the pupils are extremely small—a very important diagnostic point in a comatose patient.

Obstruction of branches of the *basilar artery* in the brain-stem produces symptoms depending on the implication of the ocular motor nuclei and the pyramidal tracts; these are described in the section on intracranial tumours (*vide infra*).

Obstruction of the *posterior inferior cerebellar artery* gives rise to a characteristic clinical picture resulting from infarction of a wedge-shaped area on the lateral aspect of the medulla. In addition to vertigo, dysphagia, signs of cerebellar deficiency, and sensory disturbances due to trigeminal involvement, there is nystagmus and Horner's syndrome (miosis, enophthalmos and ptosis) on the affected side.

Hydrocephalus. In the *congenital* and the early acquired hydrocephalus of infancy, optic atrophy is not infrequently found.

Papilloedema occurs only rarely in spite of the increased intracranial pressure, a fact doubtless due to the relief of pressure by the enlargement of the skull and the resiliency of the fontanelles and gaping sutures, as well as to the very gradual development. The eyeballs usually deviate downwards, and upward movements are restricted. Not infrequently there is considerable proptosis.

The *acquired hydrocephalus* of later life, after the fontanelles and sutures have closed, can often only be diagnosed with certainty by encephalography. The cardinal signs of increased intracranial pressure—headache, vomiting and papilloedema—are present, and to these is often added ataxia of the cerebellar type. The cases are often diagnosed as intracranial tumours in which localizing signs are not infrequently absent or masked. Bitemporal hemianopia may give a clue to the true ætiology, being due to pressure on the chiasma and tracts by the bulging floor of the third ventricle. Defective vision due to post-neuritic atrophy may persist.

INFECTIONS

Meningitis. In *acute meningococcal (epidemic) cerebrospinal meningitis* papillitis is frequently present, never papilloedema; it is due to a descending infective perineuritis. In the early stages there is often kinetic strabismus or conjugate lateral deviation of the eyes. A characteristic sign is the widely open palpebral aperture, often associated with very infrequent blinking. Paralysis of the VIth nerve, usually unilateral, is commoner than that of the IIIrd, although divergent strabismus due to the latter cause has been frequently noted. Total IIIrd nerve paralysis is rare. The pupils vary, usually showing miosis in the early stages, mydriasis when coma sets in: loss of reaction to light is relatively rare. Metastatic endophthalmitis (*q.v.*) in children is an uncommon complication.

Sporadic acute basal meningitis may be associated with complete amaurosis with normal fundi and normal pupillary reactions, pointing to the action of toxins on the higher visual centres. The blindness may persist for many weeks after subsidence of other symptoms, and sight may be completely restored. *Chronic basal meningitis* sometimes shows the same feature, but in these cases optic neuritis and post-neuritic atrophy may occur from secondary hydrocephalus and pressure of the distended third ventricle upon the chiasma and tracts or meningeal adhesions in the chiasmal region.

Purulent meningitis occurring occasionally in typhoid and more rarely in pneumonia, influenza, scarlet fever, measles, and septicæmia, may be associated with papillitis and ocular palsies.

In the meningitis of *middle ear disease* papillitis or papilloedema is usually due to complications, such as sinus thrombosis or cerebral abscess. When ocular paralysis occurs, the VIth nerve is usually affected, rarely the IIIrd (*cf. Intracranial Abscess*). The facial

nerve is most frequently involved, the paralysis often causing lagophthalmos. Conjugate deviation of the eyes is not uncommon.

Chronic chiasmal arachnoiditis is a localized meningeal infection in the meninges around the chiasma and optic nerves ; the ætiology is obscure although sepsis of the nasal sinuses or trauma may account for some cases. A primary optic atrophy usually develops bilaterally, with a central scotoma and irregular contraction of the visual fields, either concentrically or with bitemporal loss. The diagnosis from a pituitary tumour is based on negative radiological and other evidence and deformation of the chiasmal cistern on encephalography.

In *tuberculous meningitis* a moderate degree of papillitis is common (about 25 per cent.) and is generally bilateral. Papilloedema occasionally occurs and indicates the combination of solitary and miliary tubercles. In the choroid the latter are frequent and of great diagnostic importance. Until antibiotic therapy altered the almost invariably fatal prognosis of the condition, miliary choroidal tubercles were usually a terminal complication. There are often partial ocular pareses, usually of the IIIrd nerve, especially in the form of ptosis. Bilateral IIIrd paralysis is almost unknown, a point of distinction from syphilitic basal meningitis. Unilateral partial VIth nerve paralysis also occurs. Not infrequently there is a kinetic (not paralytic) conjugate deviation of the eyes and head to one side.

Brain Abscess. Cerebral abscess occurs about three times as often as cerebellar. Most cerebral abscesses are due to middle ear disease and affect the temporal lobes. Others are due to traumatism and generally affect the parietal lobes. Less common causes are metastatic infection, usually derived from the lungs, frontal sinus empyema, and orbital cellulitis. A still greater proportion of cerebellar abscesses is due to otitis media. Nearly half the cases have papilloedema, particularly cerebellar abscesses ; it is not infrequently on the side of the abscess only, and in bilateral cases the swelling is generally greater on this side. This sign has therefore greater localizing value in intracranial abscess than in tumour. Papilloedema persists longer after an operation for abscess than for tumour, or may even only then commence. As might be expected, optic atrophy is rare during the acute stage : its presence militates against the diagnosis of abscess. Ophthalmoscopic changes are more rare with extradural abscesses.

Partial unilateral IIIrd nerve paralysis is fairly common, and the combination of unilateral ptosis and mydriasis is almost pathognomonic of ipsilateral cerebral or cerebellar abscess. Partial IIIrd nerve paralysis with contralateral hemiplegia points to abscess of the temporal lobe with pressure on the IIIrd nerve and internal capsule, or more rarely to implication of the cerebral peduncle. Paralysis of the VIth nerve is not common, but is found more often in cerebellar than cerebral abscess ; it is generally ipsilateral, but

has little localizing value. Paralysis of the Vth nerve is rare. Nystagmus is common with cerebellar abscess, but rare with cerebral. In otitic cases it may be due to disease of the labyrinth.

Encephalitis. Ocular palsies usually usher in an attack of *encephalitis lethargica*. Ptosis is the commonest feature, and other branches of the IIIrd nerve are especially involved. The muscles are usually only partially paralysed and generally recover. Diplopia is an early symptom, and nystagmus may be present. Papillœdema is rare and the pupils are usually normal. The general symptoms are lethargy, with great muscular debility and other signs of an acute general infection. The disease is often followed by Parkinsonian tremor (paralysis agitans), and in the later stages spasmodic conjugate deviation of the eyes occurs (*oculogyric crises*) accompanied by synergic movements of the head and neck. Oculogyric crises may be relieved by benzedrine (up to 30 mg. a day).

Acute *polioencephalitis* accounts for the not infrequent cases of paralytic squint following a febrile attack in young children. The VIth nerve is most often involved.

SYPHILITIC INFECTIONS

Cerebral Syphilis is the term usually applied to relatively early, direct syphilitic disease of the brain and meninges. It is due essentially to gummatous inflammation of the meninges and the walls of the cerebral blood vessels.

The chief form of brain syphilis is *basal gummatous meningitis*. It usually arises in the subarachnoid tissue in the region of the chiasma and spreads thence over the base of the brain. The optic nerves, chiasma, and tracts are generally involved. Papillitis, papillœdema, or post-neuritic atrophy is frequently found (about 13 per cent. each), and is usually bilateral. Visual defects are very common, and consist of amblyopia, not infrequently amaurosis, and defects in the fields of vision. Of the latter, many cases show homonymous hemianopia from affection of one tract, fewer cases temporal hemianopia. Central scotoma and other signs of retrobulbar neuritis also occur. The IIIrd nerve is paralysed in a third of the cases, less commonly the Vth and VIth, and least frequently the IVth. The IIIrd and VIth are often affected on both sides. The trigeminal paralysis is always unilateral and often causes neuroparalytic keratitis. Pupillary changes occur, dependent upon the IIIrd nerve lesions. In many cases the process is limited to a small area, oculomotor paralysis or an affection of the visual path being the only sign except headache. A very characteristic feature of basal gummatous meningitis is the inconstancy and variability of the symptoms, temporary and recurrent visual and oculomotor disturbances being very common.

Isolated gummata may give rise to the signs of cerebral tumour, complicated by the fact that they are often multiple. Syphilitic

disease of the cerebral vessels used to be responsible for a large proportion of cases of thrombosis, hæmorrhage, softening, etc.

Tabes Dorsalis. *Syphilitic Optic Atrophy* (p. 350) occurs in about 10 to 20 per cent. of cases of locomotor ataxia. The disease is probably a primary interstitial neuritis arising as an extremely chronic exudative process from the pia and causing a secondary degeneration of the nerve fibres and their parent ganglion cells. The process usually becomes apparent first in the intracranial portion of the nerve distal to the chiasma. It is about twice as common in men as in women, most frequent between thirty and fifty years of age, and may precede the appearance of typical tabetic symptoms by some years. It is commonest in the pre-ataxic stage. The onset is gradual, leading to total blindness in two to three years or more. Pallor of the disc may precede the failure of vision by a considerable period, never the reverse. The affection of one eye usually precedes that of the other by a few months.

The *fields* show progressive contraction, *pari passu* with the failure in central vision. It is rare for the failure of sight to commence with a central scotoma, thus differing from the onset in multiple sclerosis. Two types of field are met with: (1) general concentric shrinkage, the colour fields for red and green being lost early, and central vision much impaired; (2) irregular sectorial defects, which are sharply defined but gradually spread, although central vision may be good.

The characteristic *pupillary signs* include the so-called spinal miosis, the Argyll Robertson pupil reaction, inequality of the pupils, and distortion of the pupillary aperture. These signs are found in other diseases and are to be regarded as signs of syphilis of the central nervous system rather than as pathognomonic of tabes: their combination is of great diagnostic significance. Argyll Robertson pupils are found in 70 per cent. of tabetics and are almost invariably bilateral. Unequal pupils are found in 30 per cent. of tabetics, but are met with still more frequently in general paralysis. Ophthalmoplegia interna, i.e., paralysis of the sphincter pupillæ and the ciliary muscle, occurs in about 5 per cent. of tabetics and is generally unilateral. It is due to a lesion in the nucleus of the IIIrd nerve. Cycloplegia without mydriasis, or *vice versa*, is rare.

Paralyses of the Extrinsic Ocular Muscles are common in tabes, occurring in about 20 per cent. of the cases. The order of frequency of the nerves affected is IIIrd (20 per cent.), VIth (13 per cent.), IVth (3 per cent.), external or total ophthalmoplegia (2 per cent.). It is characteristic of tabetic paralyses that they are partial pareses rather than paralyses, variable and transitory. The affection of the IIIrd nerve is always suggestive of a tabetic or syphilitic lesion. Total IIIrd nerve paralysis is rare in tabes, and isolated ptosis relatively common (4 per cent. of cases). The pareses of the ocular muscles nearly always occur in the pre-ataxic stage: when they

occur at a later stage they are more likely to be permanent. They generally clear up rapidly, but show a marked tendency to recur. Nystagmus is rare, but the paresed muscles often give rise to jerky movements of the eyes which may be mistaken for nystagmoid jerks. Paralysis of associated movements (conjugate deviations) are very rare.

General Paralysis of the Insane (*Progressive Paralysis, Paralytic Dementia*). This parasyphilitic disease is often accompanied by tabetic signs and symptoms which are due to lesions of the posterior tracts of the cord, identical with those in tabes (tabo-paralysis). The ocular symptoms are most common and unequivocal in these cases, and are to be attributed to the same causes.

The *pupillary* changes are most characteristic. In the early stages inequality is often accompanied by slight deformation in the shape of the pupil and irregularity of the pupillary margin is common. An Argyll Robertson reaction occurs in nearly half the cases. In about 5 per cent. of the cases the reactions both to light and convergence are lost, a condition which is rare in tabes and especially frequent in the juvenile form of general paralysis. The sensory reaction is very often lost with the light reaction. Spinal miosis is commoner in tabes, unequal pupils in general paralysis. Ophthalmoplegia interna is more rare in general paralysis.

Primary optic atrophy occurs in about 8 per cent. of cases showing the same type and course as in tabes. Like the pupillary signs, it may precede the onset of the typical cerebral symptoms by a considerable period, especially in those cases which commence with tabetic symptoms.

Paralysis of the extrinsic ocular muscles occur about half as frequently as in tabes, and have exactly the same characteristics, the IIIrd nerve being most frequently involved.

THE DEMYELINATING DISEASES

Multiple Sclerosis (*Disseminated or Insular Sclerosis*). Lesions in the visual paths often occur in multiple sclerosis (50 per cent. of cases). Unlike the lesions of tabes, the medullary sheaths of the nerve fibres are especially attacked, the axis cylinders remaining relatively little affected. Hence, during the acute stage, defects in conductivity are especially prominent; considerable variations succeed each other, and high degrees of functional restoration are possible. The optic nerves are most frequently attacked, with all the clinical signs of a typical retrobulbar neuritis, but patches of degeneration in the chiasma, optic tracts, or optic radiations may cause characteristic hemianopic or quadrantic changes in the fields.

The frequency of attacks of unilateral *retrobulbar neuritis* which clear up and recur, often many years before the disease becomes generalized, has already been noted (*q.v.*). They may clear up

entirely but may be followed by irregular field defects—central scotomata, concentric contraction of the field and irregular peripheral defects, sometimes only for colours, and showing variations from time to time. Some degree of optic atrophy usually develops eventually, the appearance of which bears no relation to the functional defect, but permanent blindness almost never occurs.

Nystagmus occurs in 12 per cent. of cases, but nystagmoid jerks are much commoner (50 per cent. of cases). True nystagmus is a very important diagnostic sign since it is rare in other acquired diseases of the central nervous system. Both are probably due to central changes, and the latter show some analogy to the intention tremor so characteristic of multiple sclerosis.

Miosis is fairly common in this disease, and to a less degree inequality of pupils. Other pupillary abnormalities are rare.

Paralyses of the extrinsic ocular muscles are much less common than in tabes, and although resembling these in their partial and transitory nature, differ from them in that paralyses of associated movements are not uncommon. Thus paresis of convergence, with retained action of the recti in lateral movements, is frequent; paralysis of lateral conjugate movements is commoner than that of upward and downward movements; these are due to nuclear or supranuclear lesions. Of individual nerves, the VIth is more often affected than the IIIrd, and total IIIrd nerve paralysis is never seen. Partial ophthalmoplegia externa also occurs, whereas ophthalmoplegia interna is unknown.

Neuromyelitis Optica (Devic's Disease). In this association of bilateral optic neuritis with myelitis, the visual defect usually precedes the signs of myelitis. Its onset is sudden, but one eye may be affected a day or so before the other. Complete amaurosis generally supervenes rapidly. Sometimes there is an initial central scotoma, and there may be pain on moving the eyes, pointing to a retrobulbar neuritis. There is usually only slight neuritis, but considerable swelling of the disc has been seen. In cases which recover, the blindness passes off and good vision is restored. The site of the myelitis may be lumbar or dorsal. There are no signs of general meningitis, and the other cranial nerves escape.

During the amaurotic stage the pupils are dilated and immobile. In cases of cervical and upper dorsal myelitis without optic neuritis the pupils are often unequal owing to implication of the dilatator tracts. In these cases the pupils still react to light. Unlike multiple sclerosis this type of optic neuritis does not recur.

Diffuse Sclerosis (Schilder's Disease) is characterized by a demyelination of the entire white matter of the cerebral hemispheres occurring in young people. The ocular symptoms appear early. Blindness is common, due to destruction of the optic radiations; optic neuritis or retrobulbar neuritis follows demyelination of the optic nerve. Ocular motor palsies and nystagmus are common.

HEREDITARY AND DEGENERATIVE DISEASES

Ophthalmoplegia, a primary aplasia of the ocular motor nuclei, occurs congenitally or may be acquired early in life as a hereditary familial disease ; there is usually only partial ophthalmoplegia externa, and the condition is not complicated by other nervous symptoms. *Chronic ophthalmoplegia* of a progressive type, due to a myopathy of the ocular muscles, commences with ptosis or diplopia. In the course of months or years the degeneration spreads to all the ocular muscles of both sides, except that the intrinsic muscles often escape, and not infrequently the levatores palpebrarum also. Cases of isolated ophthalmoplegia of neurogenic origin are rare, but the condition is occasionally a precursor or symptom of tabes or general paralysis of the insane, rarely of multiple sclerosis. It may become associated later with bulbar symptoms ; in these cases the internal musculature may be involved.

In **Hereditary Ataxia** (*Friedreich's Disease*) optic atrophy and paralyzes of the ocular muscles are very rare. Nystagmoid jerkings of the eyes, very similar to those occurring in multiple sclerosis, are common, but visual symptoms are absent. The movements are probably due to the same lack of co-ordination which causes the other ataxic signs of the disease ; they occur on voluntary movement, and are not usually present in passive fixation.

The **status dysraphicus** results from a defective or anomalous closure of the neural tube and may have various ocular implications. In *syringomyelia* cavities form around which secondary gliosis develops in the cervical and upper dorsal cord ; in *syringobulbia* the process extends up to the medulla. The nervous symptoms which develop between the ages of 20 and 40 include paralysis of the cervical sympathetic (Horner's syndrome), of the trigeminal and of various extra-ocular muscles. Many other ocular anomalies have been ascribed to this cause—heterochromia iridum, the retraction syndrome of Duane, and so on—but not always on sound evidence.

The **lipid dystrophies** and their associated retinal lesions, and *hepato-lenticular degeneration* with the Kayser-Fleischer corneal ring have already been described (*q.v.*).

OPHTHALMOPLÉGIA CAUSED BY DEFICIENCIES AND TOXINS

The chief exogenous poisons causing ophthalmoplegia are lead and ptomaines ; the chief toxins, diphtheria and influenza. In *lead poisoning* the onset is slow and the intrinsic muscles are often involved. In *botulism*, due to food contaminated with *Cl. botulinum*, bilateral ophthalmoplegia interna, with or without ptosis, is typical but total ophthalmoplegia also occurs. In *diphtheria* isolated ocular palsies are common, but ophthalmoplegia externa is rare ; the pupil often escapes, the ciliary muscle never. In *influenza* the palsies are similar, affecting the extrinsic and ciliary muscles, but usually not the pupil ; the pupil,

however, has been known to be affected without the ciliary muscle. In all cases recovery is common.

In *thiamine deficiency* (p. 549), frequently associated with alcoholism, the onset is sudden and accompanied by cerebral symptoms—headache, delirium, coma. Bilateral ophthalmoplegia externa occurs with or without ptosis, often followed by facial and bulbar paralyses with difficulty in speech and swallowing; the intrinsic muscles usually escape. Pathologically the condition is an *acute hæmorrhagic anterior poliomyelitis* (Wernicke). The prognosis is bad.

INTRACRANIAL TUMOURS

Intracranial tumours (including neoplasms and such lesions as tuberculomata) produce two sets of symptoms.

(1) Generalized symptoms of increased intracranial pressure—headache, vomiting, vertigo, convulsions, somnolence, alterations in the pulse, blood pressure and respiratory rhythm, papilloedema, and occasionally ocular palsies.

(2) Focal signs owing to destruction of neighbouring structures, the ophthalmologically important of which are field defects and ocular pareses.

Papilloedema has already been discussed in this relation (*q.v.*). Precentral and temporo-sphenoidal tumours are nearly always associated with severe papilloedema, post-central tumours with moderate papilloedema, often of short duration. Of subcortical tumours about one-half cause papilloedema—as a rule, moderate and of short duration. Tumours of the optic thalamus and mid-brain are almost invariably associated with papilloedema of great severity. Cerebellar tumours are constantly and extracerebellar tumours usually accompanied by papilloedema of a grave character. Of pontine tumours, only about one-half give rise to papilloedema, and then only when neighbouring parts of the brain, especially the cerebellum, have become involved: the papilloedema when it does develop is usually marked. Ventricular tumours cause a moderate papilloedema. There are two regions of the brain, the pons and the central white matter of the cerebral hemispheres, in which tumours usually develop without causing papilloedema.

Paralyses of the ocular muscles, except the lateral rectus, are rare as a sign of generalized pressure.

Focal Signs. *Prefrontal tumours*, particularly meningiomata of the olfactory groove, are sometimes associated with a pressure-atrophy of the optic nerve on the side of the lesion due to direct pressure and a papilloedema on the other side due to generalized pressure (*Foster-Kennedy syndrome*).

Chiasmal and Pituitary Tumours—see p. 355.

Tumours of the temporal lobe in 50 per cent. of cases produce a characteristic crossed upper quadrantanopia, usually incongruous,

being more accentuated in the ipsilateral field. This sign is due to pressure on the optic radiations. Visual hallucinations may occur owing to irritation of the visuo-psychic area. Downward pressure may involve the IIIrd nerve and the Vth, causing diminution of corneal sensitivity.

Tumours of the parietal lobe produce a crossed lower homonymous quadrantanopia from involvement of the upper fibres of the radiations, visual and auditory hallucinations, conjugate deviation of the eyes and optokinetic nystagmus.

Tumours of the occipital lobe present essentially visual symptoms. Typically there are crossed homonymous quadrant or hemianopic defects extending up to the fixation point. Anteriorly situated tumours may cause a crescentic loss in the periphery in the opposite uniocular temporal field. Visual agnosia may also occur.

Tumours of the mid-brain. The localizing signs of tumours in this region depend on involvement of the pyramidal tracts and ocular motor nerves. All of them may be associated with homonymous hemianopia owing to pressure on the optic tracts.

In the upper part of the mid-brain (*the colliculi and pineal gland*) the most characteristic sign is spasmodic contraction of the upper lid followed by ptosis, together with loss of conjugate movements upwards, sometimes followed by a similar failure of downward movement. In about a quarter of the cases an upper neuron facial paralysis develops in which the orbicularis escapes, and this is sometimes accompanied by an ipsilateral hemiplegia.

At an intermediate level in the *cerebral peduncles* the third nucleus becomes progressively involved. Ipsilateral ptosis and ultimately a complete IIIrd paralysis is associated with a contralateral hemiplegia involving a facial palsy of the upper neuron type (*Weber's syndrome*) (Fig. 453). If the red nucleus is involved, tremor and jerky movements occur in the contralateral side of the body: this condition, combined with ipsilateral IIIrd nerve paralysis, forms *Benedikt's syndrome*.

At a still lower level in the *pons* the same types of localizing symptoms are found. In the upper part of the pons before the fibres to the facial nucleus have crossed, there is again a IIIrd paralysis with contralateral hemiplegia and upper facial palsy. In the lower part of the pons the lateral rectus may be paralysed with a contralateral hemiplegia and an ipsilateral facial palsy (*Foville's syndrome*) (Fig. 453); more commonly the VIth palsy is replaced by a loss of conjugate movements to the same side (*Millard-Gubler's syndrome*). The Vth nerve may be paralysed causing loss of corneal sensation which is liable to cause neuroparalytic keratitis owing to the accompanying facial palsy, and implication of the VIIIth nerve may cause deafness. Miosis is rare.

Tumours of the auditory nerve, growing in the cerebello-pontine

angle, give rise to a fairly characteristic syndrome with ocular signs. Corneal anæsthesia due to implication of the Vth nerve may be an early occurrence. Early deafness on one side is associated with cerebellar symptoms, among which nystagmus is common. The VIth nerve is usually involved, generally with paralysis of the lateral rectus only, rarely with paralysis of conjugate deviation.

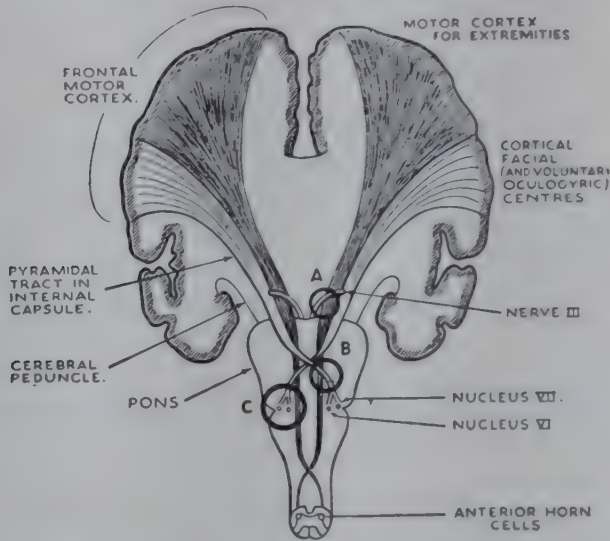


FIG. 453. The relation between the pyramidal tracts and the ocular motor nuclei. A. The site of a lesion causing Weber's syndrome (ipsilateral IIIrd paralysis and crossed hemiplegia of the face and body). B. The site of a lesion in the pons causing ipsilateral (upper neuron, i.e., without muscular atrophy) facial paralysis and contralateral hemiplegia which, on extension downwards, may involve abducens paralysis. C. The site of a lesion in the pons causing Foville's syndrome [ipsilateral abducens paralysis with loss of conjugate deviation to the same side, ipsilateral lower neuron facial palsy and contralateral (upper neuron) hemiplegia].

As might be expected, there is very often facial paralysis of the peripheral type, including the orbicularis palpebrarum.

Tumours of the cerebellum usually cause nystagmus as well as marked papilloedema.

CONGENITAL AND DEVELOPMENTAL CONDITIONS

Oxycephaly (acrocephaly) is due to precocious union of certain cranial sutures : occipito-parietal and fronto-parietal (*turriccephaly, tower skull*), or sagittal (*scaphocephaly*). Asynchronous fusion of the bones leads to a lopsided skull (*plagiocephaly*). The great wing of the sphenoid is displaced so that the orbit becomes shallow, causing some degree of proptosis. In the early stages there is papilloedema, but more commonly only the later stage of post-neuritic optic atrophy is seen. The amount of atrophy varies in degree. The papilloedema is probably due to increased intracranial pressure, owing to continued growth of the

brain in a restricted space. Most of the patients are males. Acrocephaly may be associated with syndactylism (*Apert's disease*).

Congenital Spastic Diplegia (*Little's Disease*), a bilateral spastic paralysis present from birth, considered at one time to be due to meningeal hæmorrhage as a result of birth injury, is probably a degenerative cerebral process of obscure ætiology. With it may occur such ocular anomalies as optic atrophy, retinal degeneration, cataract, squint and internuclear palsies.

HEAD INJURY

Concussion Injuries to the brain are frequently followed by hæmorrhage, usually subdural, often involving unconsciousness. The pupillary reactions are important since they may be the main diagnostic indication for operation in a patient in coma. At first the ipsilateral pupil is contracted; later, as intracranial pressure increases, this pupil dilates and does not react to light (*Hutchinson's pupil*); if pressure still increases, a similar phenomenon occurs in the other pupil. The presence of the dilated fixed pupil is a strong indication for cerebral decompression on the same side.

Fractures of the Base of the Skull commonly involve the cranial nerves. The most common complication is ipsilateral facial paralysis of the lower neuron type (22 per cent. of cases): the VIth (4 per cent.), IIIrd (2 per cent.), Vth (1·6 per cent.), and IVth (1 per cent.) follow in order of frequency. Fractures of the base sometimes involve the roof of the orbit but rarely traverse the optic foramen: occasionally both optic foramina are implicated. It may happen that the nerve is directly injured or compressed by hæmorrhage; more frequently, however, owing to the fact that the dura mater becomes the periosteum, the optic nerve is injured indirectly, probably by shearing involving laceration of the small meningeal vessels feeding it. If the injury is severe, in two to four weeks signs of primary optic atrophy appear and progress to total atrophy; in this event blindness is absolute and permanent. Papillœdema indicates hæmorrhage into the nerve sheath and may occur from basal hæmorrhage without fracture of the optic foramen; such cases have a serious prognosis. These injuries may cause concentric contraction of the field of vision, or quadrantic and other sectorial defects: a central scotoma is rare. Pigmentation in and around the disc may follow hæmorrhage into the sheath. The pupillary reactions vary and are not pathognomonic, but there is usually mydriasis on the side of the lesion.

CHAPTER 35

OCULAR MANIFESTATIONS OF SYSTEMIC DISEASES

THE most important ocular manifestations of diseases other than those of the central nervous system have already been discussed incidentally, and it will suffice here merely to enumerate them.

INFECTIVE DISEASES

Of the **bacillary infections**, *typhoid fever* causes optic neuritis, infective retinitis with hæmorrhages and occasionally metastatic choroiditis. In *whooping cough*, conjunctival, retinal and orbital hæmorrhages may occur as a result of the paroxysmal coughing. *Diphtheria* may cause a membranous conjunctivitis; it may cause cycloplegia (p. 92), and rarely paralysis of the lateral rectus. *Brucellosis*, uveitis. *Tularemia*, a nodular conjunctivitis with involvement of the proximal lymph nodes.

Leprosy causes protean ocular complications in 90 per cent. of cases—cutaneous nodules on the lids, conjunctivitis, superficial or interstitial keratitis, lepromatous lesions in the cornea and sclera, and a granulomatous uveitis, usually iritis. *Tuberculosis* affects similarly every tissue of the eye as well as being prone to excite allergic reactions. Apical phthisis may cause irritation of the sympathetic fibres, leading to dilatation of the ipsilateral pupil.

Tetanus, finding entry into a wound of the lids or globe, occasionally gives rise to the symptoms of cephalic tetanus—trismus, peripheral facial palsy and varying degrees of ophthalmoplegia.

Of the **coccal infections**, the gonococcus, the streptococcus, the staphylococcus, the pneumococcus and the meningococcus, all may give rise to metastatic implications of the eye in generalized infections, and many of them to allergic responses. Of the streptococcal infections, *erysipelas* may cause abscesses and gangrene of the lids, orbital cellulitis, and thrombosis of the orbital veins and cavernous sinus.

Scarlet fever may be accompanied by a severe conjunctivitis with a false membrane which may result in keratitis and perforation of the globe; it may also be responsible for a hypertensive retinopathy.

Of the **viral infections**, *measles* is frequently complicated by mucopurulent conjunctivitis and corneal ulcers; rarely by optic neuritis and endophthalmitis. Corneal ulcers are common in *smallpox*. *Vaccinia* of the eyelids is not uncommon and may affect the cornea, usually secondarily, sometimes causing disciform keratitis. *Chicken-pox (varicella)* occasionally shows corneal involvement. *Mumps* frequently involves a dacryoadenitis, sometimes a keratitis. *Influenza*, conjunctivitis and rarely optic neuritis or endophthalmitis; herpes of the cornea is an occasional complication. *Dengue fever*, rarely keratitis, iridocyclitis and ocular palsies. *Lymphogranuloma venereum*, corneal and conjunctival involvement. *Rubella (German measles)* in the mother during the early

months of pregnancy, particularly the second, may cause a congenital cataract in the child (*q.v.*).

Of the **Rickettsial infections**, in *typhus fever* there is severe conjunctivitis with subconjunctival hæmorrhages, corneal ulcers, occasionally iritis, optic neuritis with subsequent atrophy and ocular palsies. In *scrub typhus*, a conjunctivitis with massive subconjunctival hæmorrhages, a neuro-retinitis accompanied by hæmorrhages and exudates. *Rocky Mountain spotted fever*, a mild conjunctivitis.

Of the **spirochætal infections**, *syphilis* is protean. In *relapsing fever*, iritis, optic atrophy and ocular palsies occur; in *Weil's disease*, jaundice with conjunctivitis and subconjunctival hæmorrhages.

Fungal infections (blastomycosis, rhinosporidiosis, etc.) may invade the ocular adnexa from the surrounding tissues.

Of the **protozoan and metazoan infections**, *malaria* is associated with retinal hæmorrhages and optic neuritis. In *amœbiasis*, a conjunctivitis; in *trypanosomiasis*, a keratitis. In *Kala-azar*, retinal hæmorrhages. *Toxoplasmosis* causes a necrotizing chorioretinitis particularly in infants; sometimes a more diffuse uveitis in adults. In the former, intracranial complications sometimes involving hydrocephalus may produce indirect ocular defects. In *trichinosis*, encysted larvæ invade the extra-ocular muscles preferentially as well as the orbicularis, usually causing a characteristic oedema. Among the Cestodes, *Tænia echinococcus* may produce hydatid cysts in the eye and its adnexa, while *Tænia solium* may act similarly. *Filaria* may migrate from other parts of the body into the lids, the orbit or the globe.

A group of **muco-cutaneous inflammations** sometimes classed with *erythema multiforme* produces serious effects in the eyes; in this class, in *Stevens-Johnson's syndrome*, conjunctivitis is associated with rhinitis, stomatitis, urethritis and skin eruptions. In *Behçet's disease* a purulent uveitis with hypopyon is associated with recurrent genital or oral aphthæ. In *Reiter's disease* a severe purulent conjunctivitis and sometimes a uveitis is associated with urethritis and polyarthritides.

DISEASES OF THE CIRCULATORY SYSTEM

Pulsation of the retinal vessels, occlusion of the central artery, arteriosclerosis, and thrombosis of the central vein of the retina have already been discussed. Aneurysm at the root of the neck may cause dilatation of the pupil on the same side. In *congenital heart disease* the retinal vessels are usually dark and greatly engorged, or the veins alone may be abnormally large; retinal hæmorrhages may occur.

Degenerative arterial diseases as seen in the retina and choroid have also been discussed as well as the retinopathies associated with *hypertension*. In *Raynaud's disease*, spasmodic occlusion of the central retinal artery may occur, leading to blindness. In *Buerger's disease* (*thrombo-angiitis obliterans*), occlusion of the retinal arteries occasionally occurs. *Carotid occlusion* may result in the appearance of multiple retinal emboli.

In *giant-cell (temporal) arteritis* ophthalmic complications may be the presenting symptoms and may be of the utmost severity. These are usually due to an inflammatory destruction of the arteries to the optic nerve often leading to rapid unilateral or sometimes bilateral blindness; involvement of the central retinal artery is rare. Retinal hæmorrhages

papilloedema and diplopia are also rare. Treatment should be urgent with full doses of systemic corticosteroids and anticoagulants, but is often ineffective.

In **Diseases of the Blood**, retinal hæmorrhages, often with characteristic white exudative spots, appear in *pernicious anæmia* and also in the *secondary anæmias* of carcinomatosis, ankylostomiasis, etc. In pernicious anæmia, retrobulbar neuritis may occur. In the *purpuræ*, subconjunctival, retinal and orbital hæmorrhages. In *hæmophilia*, orbital hæmorrhages. In *polycythæmia vera*, the retinal veins are dark and tortuous.

Among the **Reticuloses** *leucæmia* is characterized by dilated retinal vessels and hæmorrhages with exudative spots. In other members of this group the lids, orbit and uveal tract may be involved with the typical lymphomatous infiltration.

Great *loss of blood* leads to amblyopia or amaurosis, and may be followed by bilateral optic atrophy, most common after uterine and intestinal hæmorrhage but rare after traumatic hæmorrhage. Although both eyes are usually affected there is often an interval of days between their involvement. The discs are hazy at first, later becoming atrophic, with constricted vessels. In some cases slight improvement of sight eventually occurs.

In *compression injuries of the chest*, venous congestion may result in conjunctival and retinal cyanosis and hæmorrhages. After fractures of the long bones, multiple fat emboli in the retina may cause hæmorrhages and the appearance of large irregular white areas (*Purtscher's traumatic angiopathic retinopathy*).

METABOLIC DISEASES

Ocular complications are common in *diabetes mellitus*, but bear little relation to the severity of the disease; they occur chiefly in long-standing cases, the most frequent being retinopathy, diabetic cataract, intra-ocular hæmorrhages, and rubeosis iridis. Œdema of the pigment epithelium on the back of the iris is often seen in microscopical specimens, but iritis is seldom present. Remarkable changes may occur in the refraction of the eye, both in the direction of hypermetropia and myopia, the former when the blood sugar falls, the latter when it rises; these are due to alterations in the refractive index of the lens, probably brought about by osmotic changes. Paralysis of both extrinsic and intrinsic ocular muscles also occur. In diabetic coma the ocular tension falls profoundly due to the osmotic extraction of fluid. Lipæmia may also occur. *Gout* has been held responsible for deposits in the conjunctiva (concretions), conjunctivitis, marginal ulcers of the cornea, episcleritis, scleritis, iritis, and other conditions; it is indirectly the cause of ocular lesions through the kidneys and vascular system. The most dramatic complication is scleritis which may be so severe as to cause retinal detachment. Night blindness is associated with some *diseases of the liver*, e.g., cirrhosis, and jaundice causes yellow discoloration of the conjunctiva, but yellow vision (*xanthopsia*) is much less common than has been thought.

The Rheumatic, Arthritic and Collagenous Diseases. *Acute rheumatism* practically never gives rise to iritis, but may cause embolism

of the central artery of the retina indirectly by its effects on the cardiac valves. It rarely causes optic neuritis. *Chronic rheumatism* is an indefinite entity, but the occurrence of iridocyclitis, episcleritis and scleritis is so frequent, particularly with rheumatoid arthritis, that there can be no doubt of the association; this is very close in children in whom a plastic iridocyclitis develops (*Still's disease*). Scleromalacia perforans is an ocular manifestation of rheumatoid nodules. In *ankylosing spondylitis* (*Marie-Strümpell's disease*) recurrent acute iridocyclitis is common. *Sjögren's syndrome* (*kerato-conjunctivitis sicca*) is also frequently associated with polyarthritis.

Muscular Diseases. In *myasthenia gravis*, ptosis occurs. In *myotonic dystrophy* ptosis may also appear; the disease is sometimes complicated by cataract, occasionally as a first symptom at an early age (30 to 40)

Deficiency Diseases are important in ophthalmology. Deficiency of vitamin A leads to xerosis of the conjunctiva, keratomalacia and night-blindness owing to interference with the re-formation of visual purple. Deficiency of vitamin B₁ (thiamine) leads to corneal and conjunctival dystrophy, severe retrobulbar neuritis with central scotomata and sometimes ophthalmoplegias. Deficiency of vitamin B₂ (riboflavine) leads to vascularizing keratitis; of vitamin C (ascorbic acid) to the conjunctival, retinal and orbital hæmorrhages and kerato-conjunctivitis of scurvy. Vitamin B₂ and C are associated with the lens metabolism and the lack of vitamin D may have some connection with cataract in rickets. Vitamin C is important in the healing of wounds of the corneal stroma.

In **Diseases of the Kidney**, hypertensive retinopathy occurs in chronic diffuse glomerulo-nephritis and in renal arteriosclerosis with renal deficiency when hypertension is present; also in the nephrosis complicating the late toxæmia of pregnancy.

Diseases of the Organs of Digestion affect the eyes mainly through focal sepsis and toxæmia arising from the teeth and throat (mainly uveitis). Infective lesions in the mouth may spread by continuity, especially along the veins of the pterygoid plexus, setting up orbital cellulitis or thrombosis of the cavernous sinus.

In association with the **Genito-urinary system** in the male, prostatic or urethral infection, sometimes accompanied by spondylitis, is a common cause of anterior uveitis; infection of the female genital tract has less association. In either sex inclusion conjunctivitis may be associated with a similar genital infection. Vague circulatory disorders (including angioneurotic oedema) have been claimed to occur in the female at the time of menstruation

SECTION VIII

PREVENTIVE OPHTHALMOLOGY

CHAPTER 36

THE CAUSES AND PREVENTION OF BLINDNESS

THE previous chapters have dealt chiefly with the diagnosis and treatment of already established diseases of the eye. An equally important branch of medical science is concerned with the prevention of disease, and although this aspect of ophthalmology has hitherto received less attention than it merits, it ought not to be ignored by the medical student or practitioner.

The most disastrous result of ocular disease, short of the relatively rare loss of life, is blindness. A study of the causes of blindness will enable the student to form a judgment as to the comparative danger of various ocular diseases.

The term "blindness" implies inability to perceive light; but it is obvious that many people who yet retain some slight degree of visual capacity are helpless from the economic standpoint. In Great Britain therefore, for practical and statutory purposes, a definition is accepted as "too blind to perform work for which eyesight is essential," and the practical limits are taken to be a visual acuity of not more than 3/60 in the better eye or alternatively a visual field reduced to a small area around the fixation point. The Register of the Blind for England, compiled on this basis, shows that there are somewhat under 100,000 blind persons.

Statistics from other countries are not readily comparable, sometimes because they are incomplete and sometimes because of differences in the assessment of blindness. Thus while in Great Britain, for purposes of registration, vision of 3/60 or less is set as a standard, in the United States and Canada the limit is 20/200. It would seem, however, on the whole, that for Western Europe, North America, Australia and the Europeanized areas in Africa and Asia, the blindness rate is of the order of 200 per 100,000 of total population. In eastern Europe, India and the South Eastern Asian States, Japan, Central and South Africa, a rate of about twice this may be assumed. In the Middle East and North Africa the rate rises to the appalling figure of 800 per 100,000 and often above, due largely to trachoma and conjunctival infections and malnutrition. As an under-estimate, statistics tend to lead to the conclusion that there are some ten million blind in the world today; a more correct figure would probably be twice or three times as high.

A sample analysis of statistics in the blind population in England and Wales shows that, of the total, approximately 60 per cent. are aged 70 years and over. The major causes are cataract, glaucoma, myopia and developmental and hereditary anomalies.

In the different age groups, different causes are operative.

(i) In infants the vast majority of cases is due to congenital anomalies, particularly cataract, optic atrophy and buphthalmos.

(ii) In school age, some 60 per cent. of cases are due to congenital and developmental anomalies, and 10 per cent. to abiotrophic defects ; a further 15 per cent. are due to neurological diseases.

(iii) In adult life (20 to 50 years) the developmental and abiotrophic defects account for 50 per cent. if myopia is included in this category ; neurological diseases account for some 20 per cent., and diabetes and trauma each 5 per cent.

(iv) In late adult life (50 to 70 years) cataract and glaucoma become common causes of blindness, each of these and myopia and diabetes contributing 15 per cent. of the total.

(v) In the elderly (70 and above) cataract and senile macular degeneration each accounts for 30 per cent., glaucoma remains at 15 per cent., while myopia, diabetes and cardio-vascular diseases each accounts for about 5 per cent.

During recent years there have been considerable changes in the incidence of the causes of blindness due largely to improvements in the control of infective conditions. This tendency has been most marked in the case of ophthalmia neonatorum, and consequently the number of blind in the early age groups has been lately much reduced. Thus the incidence of blindness was 37.0 per 100,000 school children aged 5 to 15 in 1923, and 21.3 in 1948. In 1922 the Board of Education in England, in a survey of 927 blind children in blind schools found that ophthalmia neonatorum was responsible for 30.4 per cent. of all cases of blindness ; a parallel investigation twenty-two years later showed that ophthalmia neonatorum was responsible for not more than 9.2 per cent. At the present day the disease has almost disappeared. The fall in the incidence of phlyctenular kerato-conjunctivitis, which used to account for 3.5 per cent. of blindness in children, has been equally great, a change due partly to the control of tuberculous milk and partly to better nutrition.

Retrolental fibroplasia provided an interesting interlude. In the late 1940s and early 1950s it accounted for an increasing incidence of bilateral blindness in infants, affecting the prematurely born. Since the administration of oxygen in the nursing of these children has been more strictly regulated it has virtually disappeared.

On the other hand, there is no reason for believing that any one of the four major causes of blindness—cataract, glaucoma, myopia and the congenital, hereditary and developmental anomalies—is likely to have declined to any extent. In view of the fact that the population of England is ageing, and since 84 per cent. of the blind are in the age group of 50 years and over, there seems little ground for hoping that the number of blind is decreasing as a whole. Indeed, it seems reasonable to conclude that in a developed country a further substantial decline in the number of blind in the near future is unlikely, and that the major problems of blindness now present will remain the outstanding causes. Of these, cataract, and glaucoma are diseases of the ageing; myopia and the congenital, hereditary and developmental anomalies affect the young and are essentially genetically determined. Genetic and geriatric problems relating to the two extremes of life are therefore

the essential issues outstanding in the prevention of blindness in an advanced Western community; and unfortunately we have little real knowledge nor the ability to control these conditions. Of the major causes, glaucoma is essentially a matter of early diagnosis and treatment, while cataract is one of the availability of surgery, although this is sometimes not feasible in the age group wherein its incidence is highest.

Of the other less numerically important causes, the statistics of the blind in England and Wales show the following significant factors.

(1) Syphilis is now a relatively minor cause of blindness.

(2) The inflammatory diseases are of some importance, as is shown by the fact that iritis and iridocyclitis account for 4.7 per cent. of all cases, and nondescript choroidal lesions, some of them possibly inflammatory in origin, for another 1.7 per cent.

(3) "Senile" fundus degenerations are a significant cause of blindness; "senile" macular degeneration accounts for 6.4 per cent.

(4) Vascular lesions are also of significance. Diabetes contributes 1.8 per cent. and generalized vascular diseases 1.9 per cent.

(5) Optic atrophy as an ætiologically undiagnosed group contributes 3.5 per cent., and nondescript corneal lesions 2.3 per cent. of all cases.

Industrial conditions may cause blindness, either by disease or accident. The chief diseases, such as poisoning (lead, derivatives of benzene, etc.), glass- and iron-workers' cataract, miners' nystagmus, etc., have already been discussed. Blindness due to industrial accidents is commonest among miners and in the engineering trades.

In Great Britain some indication of their frequency is given by the fact that injuries to the eyes occur in 5 per cent. of the injuries reported in factories, and in 4 per cent. of those occurring in construction work. Information on this subject is obtainable from the U.S.A. where conditions are essentially similar. It has been reliably estimated that 300,000 industrial eye accidents occur each year in that country, that is, an eye accident occurs every thirty seconds throughout every working day; every day of the working year 26,880 workers are idle because of eye injuries; each year 53,000,000 man-hours of work are lost thereby. As a result, each year approximately 1,000 American workers lose the sight of one eye irreparably, and 100 more lose the sight of both.

More striking is the enormous economic loss entailed by relatively minor accidents, such as foreign bodies in the eye. Many of these could be entirely prevented by the use of appropriate guards, screens and goggles. These matters require special attention from factory medical officers.

CHAPTER 37

THE HYGIENE OF VISION

APART from the conditions which seriously endanger the eyesight discussed briefly in the last chapter, there are many others which are liable to impair the efficiency of vision or the health of the individual. It is well known that the use of the eyes with uncorrected errors of refraction or muscle balance, or in unsuitable conditions of illumination, causes ocular pain and discomfort (commonly known as "eye-strain"), headaches, migraine, and general malaise. More serious disorders and diseases have been attributed by some to these causes. The exact pathology of "eye-strain" is unknown, and the rationale of visual fatigue in the production of ocular and systemic disorders is largely a matter of conjecture. It is, in the first place, a safe principle to correct the ametropic eye by suitable spectacles. In the next place it is necessary to study the normal limits of adaptability of the eye to various conditions of illumination, and to use the knowledge thus obtained to prevent these limits being transgressed. When we bear in mind the evolution of the visual apparatus in man, and the immense increase in the amount and nature of the work which it is called upon to perform in modern civilized life, it is a considerable compliment to the adaptability of the human body that the eyes can usually meet the demands made upon them without complaint.

Errors of Refraction. The correction of ametropia and gross errors of muscular imbalance by spectacles has already been discussed. It is evident that theoretically this correction should be made as early as possible, and especially before the increased visual strain of school life is encountered. In Great Britain much advance has been made in this direction in recent years, for the routine examination of the eyes of young school children ensures the discovery of serious errors; moreover, facilities for their correction and for the supply of suitable spectacles are now available. The most difficult problem in this connection is that of myopia, which has already been dealt with in Chapter 8. The most serious problem is the detection and treatment of amblyopia (Chapter 30) at an early stage, for which reason alone the vision of all children should be tested at the earliest age practicable.

Illumination. Normal vision is capable of adaptation to very wide ranges of intensity and quality of illumination. Form vision is very defective under dark adaptation and with low intensities of illumination. As the intensity is increased and the eye becomes light-adapted, visual acuity increases—rapidly at first, and then only very slowly. The increase is proportional to the logarithm of the intensity of the illumination, so that successive doublings or treblings of the illumination cause only equal arithmetic increments of visual acuity as estimated by the distance at which a standard letter (1.25 mm. square) can be read. Above 10 lumen/sq.ft. the increment becomes progressively less. For ordinary work this illumination suffices, but for fine work much higher

values are desirable. At extremely high illuminations, visual acuity is diminished owing to glare.

There are many factors, however, which influence visual acuity besides the intensity of the light. Among these is the size of the pupils, but more important are the amount and character of the light falling upon peripheral areas of the retina. Thus, it is undesirable that there should be too great contrast between the areas under observation and surrounding areas; for example, self-luminous figures are very difficult to focus in complete darkness, especially in conditions of fatigue, as also is a television screen in a dark room. A brilliantly illuminated field of work in an otherwise dark room causes rapid alterations of adaptation which are deleterious. Hence a moderate amount of general illumination is preferable, and this has the additional advantage that it prevents the formation of very sharply defined shadows. On the other hand, it is important that there should be no glaring lights in the field of vision; such lights should be carefully shaded. Care, too, should be taken to avoid direct reflection of light into the eyes. Thus, in reading books printed on shiny paper, and in working on bright metals, if the source of light is in front of the eyes light is reflected directly into them. This light is useless for visual purposes, and indeed, diminishes the contrasts which are the basis of discrimination. Hence the source of light should be placed laterally and somewhat behind the worker preferably, for writing, to the left-hand side in right-handed people to avoid the shadow of the hand. Flickering lights should be avoided.

Various sources of light differ much in intensity and quality. The natural criterion is sunlight which we are accustomed to regard as white light. Sunlight, however, differs much in "whiteness" and in intensity on different days, at different times of the day, and whether direct or diffuse. Owing to the adaptability of the eye it is difficult to judge the intensity of a given illumination. Measurements show that bright direct sunlight may give several thousand lumen/sq.ft., and an illumination of several hundred lumen/sq.ft. on a well-placed desk is quite common. One great advantage of daylight is its diffusion; the illumination of a room usually comes not directly from the sun, but from a considerable area of sky, and is reinforced by innumerable reflections from buildings and other objects. Sunlight is much richer in luminous radiation of short wavelength—blue and violet—than any artificial illuminants. Most modern illuminants have continuous spectra derived from incandescent solids; the higher the temperature the more nearly the energy-distribution of the spectrum approximates to that of sunlight. An approximation to diffuse daylight for purposes of matching colours, etc., can be obtained by suitable filters ("daylight lamps") and by fluorescent tubes. Incandescent gases—such as used in the mercury-vapour lamp—have line spectra; they therefore more nearly approximate monochromatic light.

Glare may be regarded as light in the wrong place. The more concentrated the light the more disturbing is the effect. Glare, therefore, varies rather with the intrinsic brilliancy of the light than with its intensity. Clear sky has a very low intrinsic brilliancy. A metal filament has an intrinsic brilliancy 350 times greater and an arc light 8,000 times higher. The ratio of the intrinsic brilliancy of a source of light to that of the surrounding field should not exceed 100.

In general, the eye works best when the object regarded is surrounded by a field illuminated to the same or slightly less degree. The illumination of the field must on no account be higher than that of the object. Glare is diminished in artificial interior illumination by the use of indirect lighting. In this method the light is reflected from the ceiling and suitably curved cornices, so that no direct light reaches the eye and shadows are almost eliminated. It is a restful but monotonous method of illumination and is quite unsuited for certain purposes. Thus, sewing is very difficult with it, especially the sewing of monochromatic material, because the threads of the texture throw no shadows, and consequently their discrimination is made very difficult. In semi-indirect lighting the use of opalescent bowls permits a certain amount of direct illumination.

TABLE OF RECOMMENDED MINIMUM SERVICE VALUES OF ILLUMINATION
FOR DIFFERENT CLASSES OF VISUAL TASK

CLASS OF VISUAL TASK	EXAMPLES	MINIMUM ILLUMINA- TION (LUX)
Casual seeing	Locker rooms	100
Rough tasks with large detail	Heavy machinery assembly; stores	200
Ordinary tasks with medium size detail	Wood machining; general offices; general assembly	400
Fairly severe tasks with small detail	Food-can inspection; clothing, cutting and sewing; business machines; drawing offices	600
Severe prolonged tasks with small detail	Fine assembly and machining; hand-tailoring; weaving silk or synthetic fibres	900
Very severe prolonged tasks with very small detail	Hosiery mending; gauging very small parts; gem cutting	1,300-2,000
Exceptionally severe tasks (minute detail)	Watchmaking; inspection of very small instruments	2,000-3,000

In domestic activities the following are recommended:

Lounges—200 lux
 Reading—400 lux
 Sewing—600 lux
 Bed-head—200 lux
 Classrooms—300; chalkboards—400

Many modern illuminants emit a considerable amount of ultra-violet radiation, which may be deleterious. Most of this is absorbed by glass, so that the dangers arising from this cause are slight and have been much over-rated. It must be remembered, however, that globes also absorb an appreciable amount of the luminous energy; even clear glass globes absorb 5-15 per cent. and opal globes as much as 10-40 per cent. The distribution of light from artificial sources varies greatly. It can be modified by the use of reflectors and prismatic (holophane) globes. Too little attention has hitherto been paid by architects and others to the position and characters of light sources from the hygienic point of

view. It is of great importance in the lighting of factories and workshops, and especially in that of schools. There has been great improvement in the lighting of schools, factories, shops, streets and houses of recent years, largely due to the work of the Illuminating Engineering Society, which has issued a schedule of Recommended Values of Illumination. The preceding table gives the general principles. On these lighting in Britain is based; American recommendations are considerably higher. The service value of illumination refers to the mean value throughout the life of the installation.

Reading and Writing. Considering the vast importance of reading and writing in modern life it is surprising that they have been so little investigated by physiologists and ophthalmologists. The forms of printed types are derived from manuscripts, and have been modified for technical reasons. Further advance has been almost entirely empirical, and even in the best presses more care has been exercised in obtaining æsthetic effects than in fostering legibility.

If we consider ordinary Roman printed characters we find that all capital letters extend above the line. Of the small letters thirteen are short, nine extend above the line (ascending letters), and five below the line (descending letters). There are thus twice as many ascending as descending letters, and in an ordinary page of print it will be found that of the long letters about 85 per cent. are ascending and only 15 per cent. descending. Examination of the short letters shows that their most characteristic features are in the upper parts. Hence, in reading, attention is specially directed to the upper parts of the letters, as is strikingly demonstrated by covering the lower parts of a line of print with a card when the print is almost as legible as if it were uncovered; if, however, the upper halves of the letters are covered, it is almost, if not quite, impossible to read the print.

The ends of the lines of which letters are composed are commonly emphasized by means of serifs. These were doubtless introduced empirically, but the advantage in sharpness of definition has a physiological basis. They counteract irradiation, and hence the visibility of letters is improved if the serifs are triangular.

The tendency of type-founders has been to minimize the differences between letters, probably with a view to attaining regularity of line and uniformity in appearance. For example, round letters have been flattened laterally and square letters rounded. The loops of b, d, p, and q, have been equalized to o. If the lower parts of short letters are covered, the similarity in the topmost curves of a, c, e, o, s, of n and r, of h and b, or of n and p, is much greater in modern print than in some early samples.

Legibility is not determined solely by visibility in the physiological sense of the term. Thus, the emphasis of some lines in letters, which originated in the use of reeds and pens for writing, increases legibility whilst diminishing visibility. A child learning to read depends upon physiological visibility; hence there should be little difference between the breadth of the thick and slender strokes. As facility in reading is acquired, legibility is increased by diminishing the breadth of the slender strokes, and as smaller letters are used the diminution must be more rapid than that of the heavy strokes, so that the interspaces may not be unduly contracted. At the same time, the slender strokes must not

transgress the limits of visibility at reading distance, and their distribution should be emphasized by suitably formed serifs. Hence, Jaeger small types are more legible than Snellen's.

The spacing of the letters and words has a considerable effect upon legibility. Here irradiation plays an important part. Roughly speaking, the interspaces between letters should be at least as broad as the blanks in m or n, but round letters like o and e should have slightly less interspace than square letters. Owing to irradiation the interspaces in general look larger than they really are, and two o's separated by a space look farther apart than two n's separated by the same space. Javal attributed a large part of the "remarkable legibility of English books" to the shortness of most English words in comparison with German and the consequent multiplication of blank interspaces. Of course, the spacing of words, and to a less degree of letters, in ordinary printing is very largely haphazard as far as legibility is concerned, the main object of the printer being to obtain general uniformity of appearance with rigid equality in the lengths of the lines. There is some difference of opinion as to whether "leading" or interlinear spacing is beneficial. Owing to the design of the blocks of type there is always a small space between the lower limits of descending and the upper limits of ascending letters, even without leading.

A line of print is read in a series of small jumps. At each pause a group of about ten letters is more or less accurately visualized; the movements are too rapid to allow visualization whilst they are occurring. The number of leaps taken by the eye remains the same irrespective of the distance of the book, so long as this is consistent with legibility. A child reading makes more jumps in a line than the average, and the same applies to people reading a foreign language or correcting proofs. Attention is directed chiefly to the commencements of words, and words are not read by letters but by their general configuration. There is, therefore, a very important psychological factor involved in the act of reading, quite apart from the interpretation of the meaning of the words.

Enough has been said to show that reading is a highly complex act, and the rules which can at present be devised for the avoidance of strain and discomfort involve a multiplicity of factors which have not yet been satisfactorily correlated.

Handicrafts. The same visual principles as have been discussed above underlie the specific problems met with in many handicrafts and industrial processes. For some types of very fine work convex lenses bringing the near point to 8 or 9 inches from the eye, combined with appropriate prisms, bases in, magnify the retinal images and have been found to give much relief.

No attempt has been made in this Section to deal exhaustively with so extensive a subject as Preventive Ophthalmology, but it has been deemed advisable to indicate to the student how innumerable and complex are the applications of ophthalmology to everyday life.

APPENDIX I

THERAPEUTIC PREPARATIONS

The strengths of lotions, etc., are given in percentages.

EYE-DROPS

In order to maintain sterility in eye-drops, the following solutions are used as solvents.

*Solution of methyl
hydroxybenzoate
(Soln. M.H.B.)*

Methyl hydroxy- benzoate . . .	0.1
Distilled water . . . to	100.0

(Not to be given over a long period.)

*Solution of phenyl-
mercuric nitrate
(Soln. P.M.N.)*

Phenylmercuric nitrate . . .	0.004
Distilled water . . . to	100.0

Adrenaline

Solution of adrenaline (1 in 1,000) . . .	10.0
Soln. M.H.B. . . to	100.0

Copper sulphate

Copper sulphate . . .	0.5
Soln. P.M.N. . . to	100.0

Amethocaine

Amethocaine hydro- chloride . . .	0.25 or 1.0
Soln. M.H.B. . . to	100.0

Cortisone

An isotonic suspension containing
0.25% w/v of cortisone acetate.

Atropine

Atropine sulphate . . .	0.125 to 2.0
Soln. M.H.B. . . to	100.0

Cortisone, strong

An isotonic suspension containing
1.0% w/v of cortisone acetate.

Bengal rose

Rose bengal (Tetra- chloro(P)tetraiodo (R)fluorescein) . . .	1.0
Soln. M.H.B. . . to	100.0

D.F.P.

Dyflor . . .	0.02 to 0.2
Sterile arachis oil . . . to	100.0

Chloramphenicol

("Chloromycetin") Chloramphenicol . . .	0.5
Boric acid . . .	2.0
Borax . . .	0.5
Soln. M.H.B. . . to	100.0

Duboisine

Duboisine sulphate . . .	0.25
Soln. P.M.N. . . to	100.0

Eserine (Physostigmine)

Eserine sulphate . . .	0.25 to 1.0
Sodium metabisulphite . . .	0.04
Soln. M.H.B. . . to	100.0

Cocaine

Cocaine hydrochloride . . .	2.0 or 4.0
Soln. M.H.B. . . to	100.0

The strength and quantity to be dispensed must be stated by the prescriber.

Ethylmorphine (Dionine)

Ethylmorphine hydro- chloride . . .	1.0 to 10.0
Soln. M.H.B. . . to	100.0

EYE-DROPS (contd.)

Fluorescein

Fluorescein sodium	2.0
Soln. P.M.N.	to 100.0

Framycetin sulphate
(“Soframycin”)

Framycetin sulphate	1.0 to 5.0
Soln. M.H.B.	to 100.0

Gelatine (Gelatine in Ringer solution)

Gelatine	0.7
Sodium chloride	0.86
Potassium chloride	0.03
Calcium chloride, anhydrous	0.024
Soln. M.H.B.	to 100.0

Glycerine

Glycerine	20.0
Soln. M.H.B.	to 100.0

Higher strengths including pure glycerine may be prescribed but should not be used without a local anæsthetic.

Homatropine

Homatropine hydrobromide	1.0
Soln. M.H.B.	to 100.0

Homatropine and cocaine

Cocaine hydrochloride	2.0
Homatropine hydrobromide	2.0
Soln. M.H.B.	to 100.0

The quantity to be dispensed must be stated by the prescriber.

Hydrocortisone

An isotonic suspension containing 0.125% w/v of hydrocortisone acetate.

Hydrocortisone and Neomycin

An isotonic suspension containing 0.125% w/v of hydrocortisone acetate and 0.5% w/v neomycin sulphate.

Hyoscine

Hyoscine hydrobromide	0.25 or 0.5
Soln. M.H.B.	to 100.0

Lachesine

Lachesine hydrochloride	1.0
Soln. M.H.B.	to 100.0

Mercurochrome

Mercurochrome	1.0
Soln. M.H.B.	to 100.0

Methylcellulose

Methylcellulose	1.0
Soln. P.M.N.	to 100.0

Neomycin

Neomycin sulphate	0.5
Soln. M.H.B.	to 100.0

Penicillin

Penicillin G	5,000 units
Sodium citrate	0.005 G.
Soln. M.H.B.	to 1.0 ml.

Phenylephrine

Asterile solution containing 2.5% or 10.0% of phenylephrine hydrochloride and a suitable bactericide.

Pilocarpine

Pilocarpine nitrate	0.5 to 4.0
Soln. M.H.B.	to 100.0

Prednisolone

An isotonic suspension containing 0.25% w/v of prednisolone acetate.

Silver protein

Silver protein B.P.	5.0, 10.0 or 20.0
Soln. M.H.B.	to 100.0

Streptomycin

Streptomycin sulphate	5,000 units
Soln. M.H.B.	to 1.0 ml.

Sulphacetamide

(“Albucid”)

Sulphacetamide sodium	10.0, 20.0 or 30.0
Boric acid	2.0
Soln. M.H.B.	to 100.0

Zinc sulphate

Zinc sulphate	0.125 or 0.25
Soln. M.H.B.	to 100.0

Zinc and adrenaline

Zinc sulphate	0.25
Solution of adrenaline (1 in 1,000)	5.0
Boric acid	2.0
Soln. M.H.B.	to 100.0

EYE LOTIONS

Ammonium tartrate

Ammonium tartrate,	
neutral . . .	10.0
Soln. P.M.N. . . to	100.0

Borax compound

Borax . . .	0.75
Sodium bicarbonate . .	0.75
Distilled water . . to	100.0

Boric acid

Boric acid . . .	2.0
Distilled water . . .	100.0

Boric acid and zinc

Boric acid . . .	2.0
Zinc sulphate . . .	0.125
Distilled water . . to	100.0

Glycerine

Glycerine . . .	12.5
Soln. M.H.B. . . to	100.0
Glycerine eye lotion is used to remove indelible pencil from the eye.	

Mercuric oxycyanide

Mercuric oxycyanide . .	0.01
Distilled water . . to	100.0

Ringer

Sodium chloride. . .	0.57
Potassium chloride . .	0.02
Calcium chloride (anhydrous) . . .	0.016
Distilled water . . to	100.0

This eye lotion is equivalent to strength Ringer solution.

Sodium bicarbonate

Sodium bicarbonate . .	2.0
Distilled water . . to	100.0

Zinc sulphate

Zinc sulphate . . .	0.125
Distilled water . . to	100.0

FOR REMOVAL OF CALCIUM DEPOSITS

Solution of sodium edetate

Di-sodium ethylenediamine tetra-acetate	0.37
Sodium bicarbonate	0.1
Distilled water to	100.0

Sterilize by autoclaving.

EYE OINTMENTS

Method of Preparation. Eye ointments are prepared by one of the following two methods :—

If the medicament is readily soluble in water, forming a stable solution, it is dissolved in the minimum quantity of sterile distilled water ; the solution is incorporated gradually with the melted base, and the mixture stirred until cold.

If the medicament is not readily soluble in water it is finely powdered, levigated with a small quantity of the base and finally incorporated with the remainder.

Sterility. Eye ointments cannot be protected by phenylmercuric nitrate or other preservative and are frequently found to carry infection if dispensed in boxes or jars. They should be dispensed only in sterile collapsible tubes.

Eye ointment basis

Liquid paraffin	10.0
Wool fat	10.0
Yellow soft paraffin	80.0

Filter while hot through a coarse filter paper in a heated funnel and sterilize by heating at 150°C for one hour.

<i>Atropine eye ointment</i>			<i>Neomycin eye ointment</i>		
Atropine sulphate .	0.5 to 2.0		Neomycin sulphate .	0.5	
Sterile eye ointment basis . . . to	100.0		Sterile eye ointment basis . . . to	100.0	
<i>Boric acid eye ointment</i>			<i>Oxytetracycline ("Tetramycin") eye ointment compound</i>		
Boric acid . . .	2.0		Oxytetracycline . . .	0.5	
Sterile eye ointment basis . . . to	100.0		Bacitracin . . .	0.1	
<i>Chloramphenicol ("Chloromycetin") eye ointment</i>			Sterile basis . . . to	100.0	
Chloramphenicol .	1.0		<i>Penicillin eye ointment</i>		
Sterile eye ointment basis . . . to	100.0		Penicillin G . . .	5,000 units	
<i>Chlortetracycline ("Aureomycin") eye ointment</i>			Sterile eye ointment basis . . . to	1.0 G.	
Chlortetracycline .	1.0		<i>Pilocarpine eye ointment</i>		
Sterile eye ointment basis . . . to	100.0		Pilocarpine nitrate .	1.0 or 2.0	
<i>Cortisone eye ointment</i>			Sterile eye ointment basis . . . to	100.0	
Cortisone acetate .	0.5		<i>Polymyxin and Bacitracin eye ointment ("Polyfax")</i>		
Sterile eye ointment basis . . . to	100.0		Polymyxin B sulphate	10,000 units	
<i>Eserine (Physostigmine) eye ointment</i>			Bacitracin . . .	500 units	
Eserine salicylate .	0.25 or 0.5		Sterile basis . . . to	1.0 G.	
Sterile eye ointment basis . . . to	100.0		<i>Prednisolone eye ointment</i>		
<i>Hydrocortisone eye ointment</i>			Prednisolone acetate .	0.25	
Hydrocortisone acetate	0.5		Sterile eye ointment basis . . . to	100.0	
Sterile eye ointment basis . . . to	100.0		<i>Streptomycin eye ointment</i>		
<i>Hydrocortisone and Neomycin eye ointment</i>			Streptomycin sulphate	5,000 units	
Hydrocortisone acetate	0.125		Sterile eye ointment basis . . . to	1.0 G.	
Neomycin sulphate .	0.5		<i>Sulphacetamide ("Albucid") eye ointment</i>		
Sterile eye ointment basis . . . to	100.0		Sulphacetamide sodium . . .	5.0	
<i>Hyoscine eye ointment</i>			Sterile eye ointment basis . . . to	100.0	
Hyoscine hydrobromide	0.5		<i>Tetracycline ("Achromycin") eye ointment</i>		
Sterile eye ointment basis . . . to	100.0		Tetracycline . . .	1.0	
<i>Mercuric oxide eye ointment</i>			Sterile basis . . . to	100.0	
Yellow mercuric oxide, freshly precipitated .	0.5 or 1.0				
Sterile eye ointment basis . . . to	100.0				

ARTIFICIAL TEARS

Serum (human or horse)	10.0
Sodium chloride	0.774
Potassium chloride	0.027
Calcium chloride, anhydrous	0.022
Chlorbutol	0.3
Distilled water to	100.0

CAUSTIC

Iodizing solution (for dendritic ulcers, etc.)

Iodine	7.0
Potassium iodide	5.0
Alcohol 90% to	100.0

INJECTIONS

FOR ANTERIOR CHAMBER

Acetylcholine injection

Acetylcholine	0.01
In Ringer's soln.	

Anterior chamber irrigation

Sodium chloride	0.86
Potassium chloride	0.03
Calcium chloride, anhydrous	0.024
Distilled water to	100.0

Sterilize by autoclaving. This is Ringer Solution.

Pilocarpine injection (isotonic)

Pilocarpine nitrate	0.5
Sodium chloride	0.57
Potassium chloride	0.02
Calcium chloride, anhydrous	0.016
Water for injection B.P. . . . to	100.0

The injection in 1 ml. ampoules is sterilized by autoclaving.

RETROBULBAR INJECTIONS

All retrobulbar injections should be preceded by an injection of procaine 2%.

Alcohol injection

Alcohol 40% v/v; 70% v/v or 90% v/v.

Sterilized by autoclaving in sealed ampoules.

Tolazoline ("Priscol") injection

Tolazoline hydrochloride 25 mg. in 1 ml.

FOR LOCAL ANÆSTHESIA AND AKINESIA

Lignocaine hydrochloride injection

Lignocaine hydrochloride	2.0
Water for injection to	100.0

Lignocaine and adrenaline injection

Lignocaine hydrochloride	2.0
Sodium chloride	0.45
Solution of adrenaline (1 in 1,000)	1.25
Sodium metabisulphite	0.1
Water for injection to	100.0

Procaine injection

Procaine hydrochloride	2.0
Sodium metabisulphite	0.1
Chlorocresol	0.1
Water for injection to	100.0

Procaine and adrenaline injection

Procaine hydrochloride	2.0
Solution of adrenaline (1 in 1,000)	2.0
Sodium metabisulphite	0.1
Chlorocresol	0.1
Water for injection to	100.0

Procaine and alcohol injection

Procaine hydrochloride	4.0
Alcohol 90%	16.66
Sodium metabisulphite	0.1
Chlorocresol	0.1
Water for injection to	100.0

SUBCONJUNCTIVAL INJECTIONS

For children: Mydricaine

No. 1

Atropine sulphate . . .	0.166
Procaine hydrochloride . . .	1.0
Solution of adrenaline (1-1,000) . . .	20.0
Boric acid . . .	1.76
Sodium metabisulphite . . .	0.1
Water for injection to	100.0

Dose not to exceed 0.3 ml. which contains atropine, procaine and adrenaline in the following proportions:—

0.5 mg., 3.0 mg., 0.06 ml.

For adults: Mydricaine

No. 2*

Atropine sulphate . . .	0.333
Procaine hydrochloride . . .	2.0
Solution of adrenaline (1-1,000) . . .	40.0
Boric acid . . .	1.76
Sodium metabisulphite . . .	0.1
Water for injection to	100.0

Dose not to exceed 0.3 ml. which contains atropine, procaine and adrenaline in the following proportions:—

1.0 mg., 6.0 mg., 0.12 ml.

**Caution*

A 0.3 ml. dose of this solution contains 1.0 mg. of atropine sulphate which is the maximum dose stated in the British Pharmacopœia.

Hyaluronidase injection

Available as a dry powder in ampoules containing the equivalent of 1,000 units.

This powder is dissolved by the addition of sterile distilled water or other drug to be used in combination; e.g., cortisone suspension.

The solution is unstable beyond twenty-four hours.

Subconjunctival injection of penicillin

Penicillin G	500,000 units
Lignocaine and adrenaline injection to	0.5 ml.

Subconjunctival injection of polymyxin

Polymyxin B sulphate	125,000 units
Lignocaine and adrenaline injection to	0.5 ml.

Pain is usually severe and prolonged following this injection and necessitates adequate analgesia.

Subconjunctival injection of "Soframycin"

Framycetin sulphate ("Soframycin") . . .	500 mg.
Distilled water . . .	1.0 ml.

LAMELLÆ

Lamellæ atropinæ

Each disc contains 0.13 mg. of atropine sulphate.

Lamellæ cocainæ

Each disc contains 0.65 mg. of cocaine hydrochloride.

For use as an anæsthetic when drops are not available.

The strength and quantity to be dispensed must be stated by the prescriber.

Lamellæ homatropinæ

Each disc contains 0.65 mg. of homatropine hydrobromide.

Lamellæ hyoscine

Each disc contains 0.26 mg. of hyoscine hydrobromide.

Lamellæ physostigminæ

Each disc contains 0.65 mg. of physostigmine salicylate.

PAINTS

Brilliant green and crystal violet paint (aqueous)

Brilliant green . . .	0.5
Crystal violet . . .	0.5
Distilled water . . . to	100.0

Silver nitrate paint

Silver nitrate . . .	1.0 or 2.0
Distilled water . . . to	100.0
This preparation should be protected from light by storage in amber-coloured bottles fitted with ground glass stoppers to prevent evaporation	

THE APPLICATION OF HEAT TO THE EYE

Heat may be applied to the eye moist or dry. The best method for the application of **moist heat** is that of *hot bathing*. A pad of cotton wool is tied into the bowl of a wooden spoon. The wool is dipped into a bowl of hot water which can be replenished, and is then approximated to the closed eye. As soon as it has cooled sufficiently it is brought into contact with the closed lids. As soon as it ceases to feel hot the wool is again dipped in the hot water and the process repeated. The bathing is continued for ten to fifteen minutes, and then a pad of dry warm cotton wool is bandaged over the eye. The hot bathings may be repeated frequently.

An alternative is the application of *hot compresses*. These are large round pads of plain or boric lint, on one surface of which gutta-percha tissue is sewn. The compresses are placed in a cloth and immersed in boiling water; by keeping the ends of the cloth out of the water and turning them in opposite directions the excess of the water is wrung out without scalding the fingers. The compress is applied as hot as can be borne. It is at once covered with a large pad of hot cotton wool and bandaged into position.

Dry heat may be applied over long periods by means of an *electric eye pad*, an eye pad of gauze heated by incorporating in it a small electric resistance. An alternative method is by *medical diathermy*. An eye pad, composed of layers of cotton wool wrung out in warm saline and applied evenly to the closed lids, serves as one of the electrodes, the other being bound to the arm. The current is slowly increased until the desired amount of heat is attained. This is generally between 300 and 600 milliamperes: it is maintained at this reading for five minutes and then slowly reduced to zero.

VITAMINS

The following are the vitamins most important in ophthalmology, showing their natural sources, daily need, functions, and deficiency effects.

A. (Higher alcohol synthesized from carotene in the liver). *Carotene* from carrots, green vegetables; **A** from fat of fish, esp. liver—*cod liver oil, halibut oil*—egg yolk, milk: *carotene* 3 mgms; **A**. 3,000 units: maintenance of healthy ectodermal structures (respiratory, alimentary, urinary, conjunctival, corneal, retinal); *production of visual purple*. Deficiency effects—dermatoses, demyelination, diminished resistance to infections; *xerosis, xerophthalmia, keratomalacia, night blindness*.

B₁ (Thiamine, a pyrimidine thiazole compound). From many foodstuffs, esp. lean pork, beans, peas, nuts, whole grain and flour, beef, yeast:

1 mgm. : carbohydrate metabolism. Deficiency effects—beri-beri, peripheral neuritis ; *corneal and conjunctival dystrophy, retrobulbar neuritis.*

B₂ or G. (Riboflavine). Same sources as B₁ : 1 mgm. : oxygenation. Deficiency effects—glossitis and cheilosis ; *vascularizing keratitis.*

Several other vitamins of the B group are now known but their precise importance is not yet fully determined.

C. (Ascorbic or cevitamic acid). From fresh fruit and vegetables (destroyed by heating) : 50 mgms. : Blood formation, osteogenesis, *lens metabolism.* Deficiency effects—scurvy, anæmia, osteoplasia ; *conjunctival and retinal hæmorrhages, kerato-conjunctivitis.*

D. (Calciferol—isomer of ergosterol, formed by ultra-violet light on skin). From animal fats, esp. *cod liver oil and halibut oil* ; sunshine : 1,000 units, 0.025 mgm. : Ca and P metabolism. Deficiency effects—rickets, osteomalacia, dental caries, tetany ; *cataract.*

K. (Dimethylnaphthoquinone). From alfalfa : prothrombin formation.

P. (Flavone). From citrous fruits : maintenance of health of capillaries. Deficiency effects of vitamins K and P—hæmorrhagic conditions.

These vitamins have been isolated or synthesized, and are available in proprietary preparations. Deficiency should be counteracted as far as possible in the diet. Little harm seems to accrue from large doses, except in the case of hypercalcæmia from calciferol.

CARE OF INSTRUMENTS

Ophthalmic instruments should be kept in an air-tight glass cabinet, or when not constantly in use in velvet-lined cases.

All instruments may safely be sterilized before use by boiling in 3 per cent. sodium carbonate solution (*not* bicarbonate), made with *distilled* water. This procedure does not impair the cutting edges, but knives and scissors should not be boiled more than three to five minutes : this is amply sufficient if the surfaces are bright and free from tarnish, as they ought to be. If distilled water cannot be obtained the cutting instruments should be well soaked in pure carbolic acid and then washed in sterile saline before use.

The instruments should be removed from the sterilizer *immediately* before operating and used dry. *In no case must instruments be immersed in boric lotion, since it tarnishes the steel.*

It is safer to use the instruments dry as it is almost impossible to sterilize the skin of the hands efficiently, and if the instruments are wet, fluid from the fingers is liable to run along them into the eye. Dry sterilization in a hot air oven offers many advantages—efficiency, the avoidance of damage to sharp cutting edges, and the possibility of the surgeon taking the instruments directly from the container in which they have been sterilized without handling by any other person. The points of knives should be dipped in sterile saline immediately before use to facilitate their passage through the tissues. While dry sterilization is the best technique, its disadvantage is the time which it takes (1½ to 2 hours) ; for this reason in a busy hospital a separate set of instruments is required for each operation undertaken in a half-day.

The surgeon should wear a sterilized gown and also a mask containing a layer of cellophane covering the nose and mouth for all operations, as well as sterilized gloves whenever there is the possibility of the fingers touching the surgical area.

APPENDIX II

VISUAL ACUITY TRANSCRIPTION TABLES

For some decimals the distance at the examination has to be altered from 6 to 5 m. or from 20 to 15 feet.

Decimal system	Snellen 6-metre table	20-foot table	Resolution angle table
1·0	6/6	20/20	1·0
0·8	5/6	20/25	1·3
0·7	6/9	20/30	1·4
0·6	5/9	15/25	1·6
0·5	6/12	20/40	2·0
0·4	5/12	20/50	2·5
0·3	6/18	20/70	3·3
0·1	6/60	20/200	10·0

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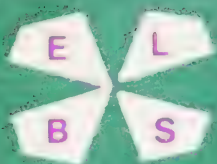
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